Pharmacology: Gastro, Fluids and MSK

Question 1 of 49

Regarding ibuprofen, which of the following is CORRECT:

a It has greater efficacy than naproxen.

b It has more side effects than naproxen.

c It is similar in efficacy to naproxen.

d It has a similar side effect profile to naproxen.

e It has a lower efficacy but less side effects than naproxen.

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Pharmacology: Gastro, Fluids and MSK

Question 1 of 49

Regarding ibuprofen, which of the following is CORRECT:

a) **It has greater efficacy than naproxen.** ✗
b) It has more side effects than naproxen.
c) It is similar in efficacy to naproxen.
d) It has a similar side effect profile to naproxen.
e) It has a lower efficacy but less side effects than naproxen. ✓

Answer

Ibuprofen has fewer side effects than other non-selective NSAIDs but its anti-inflammatory properties are weaker, making it unsuitable for conditions where inflammation is prominent e.g. acute gout. Naproxen has good efficacy with a relatively low incidence of side effects (but more than ibuprofen); it is recommended first line treatment in acute gout.

Notes

Mechanism of action

NSAIDs reduce the production of prostaglandins by inhibiting the cyclo-oxygenase (COX) enzymes with analgesic, antipyretic and anti-inflammatory effects. The two main types of COX enzyme are COX-1 (which produces prostaglandins that help to maintain gastric mucosal integrity and platelet-initiated blood clotting) and COX-2 (which produces prostaglandins that mediate pain and inflammation). NSAIDs vary in their selectivity for inhibiting different types of COX; specific inhibition of COX-2 (e.g. celecoxib and etoricoxib) is associated with less gastrointestinal intolerance.

Therapeutic effects

In single doses NSAIDs have analgesic activity comparable to that of paracetamol, therefore given their side effect profile, paracetamol is preferred, particularly in the elderly. In regular full dosage NSAIDs have both a lasting analgesic and an anti-inflammatory effect making them useful for the management of continuous or regular pain associated with inflammation e.g. inflammatory arthritides. Pain relief starts soon after taking the first dose and a full analgesic effect should normally be obtained within a week, whereas an anti-inflammatory effect may not be achieved (or may not be clinically assessable) for up to 3 weeks.

Choice of NSAID

Ibuprofen has fewer side effects than other non-selective NSAIDs but its anti-inflammatory properties are weaker, making it unsuitable for conditions where inflammation is prominent e.g. acute gout.

Naproxen has good efficacy with a relatively low incidence of side effects (but more than ibuprofen); it is recommended first line treatment in acute gout.

Diclofenac is similar in efficacy to naproxen but is associated with a higher incidence of cardiovascular adverse events (and is in fact contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure).

Mefenamic acid has only minor anti-inflammatory properties and is typically used in the management of painful dysmenorrhoea. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment.

Risk of NSAID-induced GI adverse events	NSAIDs
Lowest risk	Ibuprofen
Intermediate risk	Naproxen, diclofenac, indometacin
Highest risk	Piroxicam, ketoprofen, ketorolac

Contraindications

NSAIDs are contraindicated in people with:

- A history of hypersensitivity or severe allergic reaction to aspirin or any other NSAID
- Severe heart failure (NSAIDs may impair renal function)
- Liver fibrosis, cirrhosis or acute liver failure (risk of variceal bleeding, hepatorenal syndrome and encephalopathy)
- Severe hepatic impairment (e.g. liver enzyme levels more than three times the upper limit of the normal range; serum albumin less than 25 g/L)
- Current treatment for gastrointestinal bleeding, symptomatic peptic ulcer, or gastrointestinal perforation or obstruction
- Coxibs and diclofenac are also contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, mild, moderate, or severe heart failure
- Renal failure, with estimated glomerular filtration rate (eGFR) less than 30–15 mL/min/1.73 m2, or creatinine clearance less than 30–20 mL/min, or dehydration (relative)

Cautions

NSAIDs should be used with caution in:

- People with asthma (patients should be offered a trial to assess effect on asthma control)
- The elderly (increased risk of serious adverse effects such as gastrointestinal bleeding and perforation, which may be fatal)
- People with a history of peptic ulceration (standard NSAIDs contraindicated), or those at high risk of gastrointestinal adverse effects
- People with inflammatory bowel disease (NSAIDs may increase the risk of developing or cause exacerbations of ulcerative colitis or Crohn's disease)
- People with hepatitis or cholestasis (increased risk of gastrointestinal bleeding and fluid retention)
- Renal impairment (avoid if possible, sodium and water retention may occur leading to a deterioration in renal function and, possibly renal failure)
- People with heart failure (NSAIDs may impair renal function)
- People with hypertension (NSAIDs may impair renal function)
- In a women trying to conceive (NSAIDs may impair female fertility)

Adverse effects

Adverse effects may be minimised by selecting an appropriate NSAID and using the lowest effective dose for the shortest duration necessary to control symptoms.

Side effects of NSAIDs include:

- The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications such as ulcer, perforation, obstruction or bleeding (especially if risk factors are present – see below)
- Less commonly NSAIDs may cause cardiovascular and renal complications such as myocardial infarction, stroke, cardiac failure, hypertension, and renal failure (the risk is increased in people with IHD, CVD, PAD, CKD, HF or HTN, risk factors for cardiovascular disease and people > 65 years)
- Prolonged bleeding (for example after surgery) because of platelet inhibition
- Asthma (NSAIDs may exacerbate or precipitate asthma. Stop NSAID if it is suspected to have precipitated bronchospasm)
- Severe skin reactions and angioedema (stop NSAID)
- Very rarely, NSAIDs can precipitate severe hepatic reactions such as hepatitis, liver necrosis, or hepatic failure (stop NSAID)

Risk factors for increased risk of gastrointestinal adverse effects

People are at high risk of serious NSAID–induced gastrointestinal adverse events if they have one or more of the following risk factors:

- Using the maximum recommended dose of an NSAID
- Aged 65 years or older
- History of gastroduodenal ulcer, GI bleeding, or gastroduodenal perforation
- Concomitant use of medications that are known to increase the likelihood of upper GI adverse events (e.g. anticoagulants, aspirin, corticosteroids, and antidepressants (selective serotonin reuptake inhibitors, venlafaxine, or duloxetine))
- Serious comorbidity, such as cardiovascular disease, hepatic or renal impairment, diabetes, or hypertension
- Requirement for prolonged NSAID use including people with osteoarthritis or rheumatoid arthritis of any age and people aged 45 years or older with chronic low back pain
- The presence of Helicobacter pylori infection
- Excessive alcohol use
- Heavy smoking

Prevention and management of gastrointestinal adverse effects

- Preventing gastrointestinal adverse effects
  - Consider prescribing:
    - An alternative to an NSAID e.g. paracetamol +/- codeine
    - The lowest dose of NSAID for the shortest period of time
    - Only one NSAID at a time (avoid concomitant use with aspirin)
    - A COX-2 selective NSAID (but increased cardiovascular risk)
  - If the person is at an increased risk of GI adverse events and requires an NSAID, co-prescribe a proton pump inhibitor (PPI) for gastroprotection (e.g. omeprazole 20 mg od, lansoprazole 15 – 30 mg od)
- Managing gastrointestinal adverse effects
  - Where possible the NSAID should be withdrawn if an ulcer occurs
  - The ulcer should be treated with a PPI (a H2-receptor antagonist or misoprostol are alternatives)
  - On healing the patient should be tested for H. pylori and treated if required
  - If treatment with NSAID needs to continue, options include:
    - On healing continue PPI
    - Switch to selective COX-2 inhibitor

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Pharmacology: Gastro, Fluids and MSK

Question 2 of 49

A 34 year old overweight man, who has a history of gout, presents to ED with an acutely exquisitely tender big toe. His big toe is red, hot and swollen, but he is apyrexial and otherwise systemically well. Acute gout is diagnosed. What is the most appropriate first line treatment for this patient:

a Allopurinol

b NSAIDs

c Colchicine

d Oral corticosteroids

e Intraarticular corticosteroids

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Pharmacology: Gastro, Fluids and MSK

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a) **Allopurinol** ✗
b) NSAIDs ✓
c) Colchicine
d) Oral corticosteroids
e) Intraarticular corticosteroids

Answer

NSAIDs (such as diclofenac, indometacin or naproxen) are usually the first line treatment for acute gout. Colchicine is an alternative in patients in whom NSAIDs are contraindicated, not tolerated or ineffective. Oral or parenteral corticosteroids are an effective alternative in those who cannot tolerate or who are resistant to NSAIDs and colchicine. Intra-articular injection of a corticosteroid can be used occasionally in acute monoarticular gout. Allopurinol is not a treatment for acute gout (but should be continued during an acute attack if the patient is already established on long term therapy).

Notes

Gout is a disorder of purine metabolism characterised by a raised uric acid level in the blood (hyperuricaemia) and the deposition of urate crystals in joints and other tissues, such as soft connective tissues or the urinary tract.

NSAIDs

Acute attacks of gout are usually treated with an NSAID such as diclofenac, indometacin, or naproxen. Treatment should be started as soon as possible and continued until 48 hours after the attack has resolved. A PPI should be co-prescribed for gastric protection in people at high risk of gastrointestinal adverse events.

Colchicine

Colchicine is an alternative in patients in whom NSAIDs are contraindicated, not tolerated or ineffective. Although its use is limited by toxicity at higher doses, it is useful in patients with heart failure or those taking anticoagulants.

The dose is 500 micrograms 2 – 4 times a day until the symptoms are relieved or diarrhoea or vomiting occurs (max 6 mg per course and do not repeat treatment within 3 days).

Colchicine should be avoided in people with blood dyscrasias and bone marrow disease.

Common side effects of colchicine include:

- abdominal pain
- nausea and vomiting
- diarrhoea

Side effects are dose dependent and may be severe enough to limit treatment.

Corticosteroids

Oral or parenteral corticosteroids are an effective alternative in those who cannot tolerate or who are resistant to NSAIDs and colchicine. Intra-articular injection of a corticosteroid can be used occasionally in acute monoarticular gout.

Allopurinol

Allopurinol is not effective in treating an acute attack and may prolong it indefinitely if started during the acute episode, but should be continued during an acute attack if the patient is already established on long term therapy.

Long term treatment (prevention) of gout may be indicated in patients with frequent recurrence of acute attacks of gout, the presence of tophi, or signs of chronic gouty arthritis. Allopurinol reduces uric acid formation from purines and may be used to prevent further attacks of gout by correcting hyperuricaemia. The initiation of treatment may precipitate an acute attack, and should be covered with an NSAID or colchicine, continued for at least one month after the hyperuricaemia has been corrected.

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Pharmacology: Gastro, Fluids and MSK

Question 3 of 49

Normal saline (sodium chloride 0.9%) contains how much sodium:

a 131 mmol/L

b 150 mmol/L

c 111 mmol/L

d 170 mmol/L

e 126 mmol/L

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Pharmacology: Gastro, Fluids and MSK

Question 3 of 49

Normal saline (sodium chloride 0.9%) contains how much sodium:

a) 131 mmol/L
b) 150 mmol/L ✔
c) **111 mmol/L** ✖
d) 170 mmol/L
e) 126 mmol/L

Answer

Normal saline (sodium chloride 0.9%) contains:

- Na^+ 150 mmol/L
- Cl^- 150 mmol/L

Notes

Solutions of electrolytes are given intravenously to meet normal fluid and electrolyte requirements or to replenish substantial deficits or continuing losses when the patient is nauseated or vomiting and is unable to take adequate amounts by mouth. When intravenous administration is not possible, fluid (as sodium chloride 0.9% or glucose 5%) can also be given by subcutaneous infusion.

Sodium chloride

Normal saline (sodium chloride 0.9%) contains:

- Na^+ 150 mmol/L
- Cl^- 150 mmol/L

Sodium chloride in isotonic solution (0.9%) provides the most important extracellular ions in near physiological concentrations and is indicated in sodium depletion which can arise from such conditions as gastroenteritis, diabetic ketoacidosis, ileus, and ascites.

Chronic hyponatraemia arising from inappropriate secretion of antidiuretic hormone should ideally be corrected by fluid restriction. If sodium chloride is required for acute or chronic hyponatraemia, regardless of the cause, the deficit should be corrected slowly to avoid the risk of osmotic demyelination syndrome and the rise in plasma sodium concentration should not exceed 10 mmol/L in 24 hours. In severe hyponatraemia, sodium chloride 1.8 % may be used cautiously.

Hartmann's solution

Hartmann's solution (compound sodium lactate) can be used instead of isotonic sodium chloride solution during or after surgery, or in the initial management of the injured or wounded; it may reduce the risk of hyperchloraemic acidosis.

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
- HCO_3^- 29 mmol/L
- Cl^- 111 mmol/L
- Ca^{2+} 2 mmol/L

Glucose

Glucose solutions (5%) are used mainly to replace water deficit. Average water requirements in a healthy adult are 1.5 to 2.5 litres daily and this is needed to balance unavoidable losses of water through the skin and lungs and to provide sufficient for urinary excretion. Dehydration may occur when these losses are not compensated for by intake e.g. in coma, or in the elderly. Excessive loss of water without loss of electrolyte is uncommon, occurring in fevers, hyperthyroidism, diabetes insipidus and hypercalcaemia.

Glucose solutions are also used to correct and prevent hypoglycaemia and to provide a source of energy in those too ill to be fed adequately by mouth.

Glucose solutions are also given in regimens with calcium and insulin for the emergency management of hyperkalaemia. They are also given after correction of hyperglycaemia during treatment of diabetic ketoacidosis, when they must be accompanied by continuing insulin infusion.

Potassium chloride mixtures

Potassium chloride with sodium chloride intravenous infusion is the initial treatment for the correction of severe hypokalaemia and when sufficient potassium cannot be taken by mouth. Repeated measurement of plasma-potassium concentration is necessary to avoid the development of hyperkalaemia, particularly in renal impairment. Initial potassium replacement therapy should not involve glucose infusions, because glucose may cause a further decrease in the plasma-potassium concentration.

Sodium bicarbonate

Sodium bicarbonate is used to control severe metabolic acidosis (pH < 7.1) particularly that caused by loss of bicarbonate (as in renal tubular acidosis or from excessive gastrointestinal losses). Mild metabolic acidosis associated with volume depletion should first be managed by appropriate fluid replacement because acidosis usually resolves as tissue and renal perfusion are restored.

In more severe metabolic acidosis or when the acidosis remains unresponsive to correction of anoxia or hypovolaemia, sodium bicarbonate (1.26%) can be infused over 3 – 4 hours with plasma-pH and electrolyte monitoring. In severe shock, for example in cardiac arrest, metabolic acidosis can develop without sodium or volume depletion; in these circumstances sodium bicarbonate is best given as a small volume of hypertonic solution, such as 50 mL of 8.4% solution intravenously.

Albumin

Albumin solutions, prepared from whole blood, contain soluble proteins and electrolytes but no clotting factors, blood group antibodies, or plasma cholinesterases thus may be given without regard to the recipient's blood group.

Albumin is usually used after the acute phase of illness to correct a plasma-volume deficit; hypoalbuminaemia itself is not an appropriate indication and the use of albumin solution in acute plasma or blood loss is wasteful where plasma substitutes should be used instead.

Concentrated albumin solution (20%) can be used under specialist supervision in patients with an intravascular fluid deficit and oedema because of interstitial fluid overload, to restore intravascular plasma volume with less exacerbation of the salt and water overload than isotonic solutions. Concentrated albumin solution may also be used to obtain a diuresis in hypoalbuminaemic patients (e.g. in hepatic cirrhosis).

Plasma substitutes

Dextran and gelatin may be used at the outset to expand and maintain blood volume in shock arising from conditions such as burns or septicaemia; they may also be used as an immediate short-term measure to treat haemorrhage until blood is available. Dextran and gelatin are rarely needed when shock is due to sodium and water depletion because, in these circumstances, the shock responds to water and electrolyte repletion.

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Pharmacology: Gastro, Fluids and MSK

Question 4 of 49

NSAIDs are contraindicated in all of the following conditions EXCEPT for:

a History of hypersensitivity to aspirin

b Liver cirrhosis

c Active peptic ulcer disease

d Inflammatory bowel disease

e Severe heart failure

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Pharmacology: Gastro, Fluids and MSK

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NSAIDs are contraindicated in all of the following conditions EXCEPT for:

a) **History of hypersensitivity to aspirin** ✗
b) Liver cirrhosis
c) Active peptic ulcer disease
d) Inflammatory bowel disease ✓
e) Severe heart failure

Answer

NSAIDs should be used with caution in inflammatory bowel disease as they may increase the risk of developing or cause exacerbations of ulcerative colitis or Crohn's disease.

NSAIDs are contraindicated in people with:

- A history of hypersensitivity or severe allergic reaction to aspirin or any other NSAID
- Severe heart failure (NSAIDs may impair renal function)
- Liver fibrosis, cirrhosis or acute liver failure (risk of variceal bleeding, hepatorenal syndrome and encephalopathy)
- Severe hepatic impairment (e.g. liver enzyme levels more than three times the upper limit of the normal range; serum albumin less than 25 g/L)
- Current treatment for gastrointestinal bleeding, symptomatic peptic ulcer, or gastrointestinal perforation or obstruction
- Coxibs and diclofenac are also contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, mild, moderate, or severe heart failure
- Renal failure, with estimated glomerular filtration rate (eGFR) less than 30–15 mL/min/1.73 m2, or creatinine clearance less than 30–20 mL/min, or dehydration (relative)

Notes

Mechanism of action

NSAIDs reduce the production of prostaglandins by inhibiting the cyclo-oxygenase (COX) enzymes with analgesic, antipyretic and anti-inflammatory effects. The two main types of COX enzyme are COX-1 (which produces prostaglandins that help to maintain gastric mucosal integrity and platelet-initiated blood clotting) and COX-2 (which produces prostaglandins that mediate pain and inflammation). NSAIDs vary in their selectivity for inhibiting different types of COX; specific inhibition of COX-2 (e.g. celecoxib and etoricoxib) is associated with less gastrointestinal intolerance.

Therapeutic effects

In single doses NSAIDs have analgesic activity comparable to that of paracetamol, therefore given their side effect profile, paracetamol is preferred, particularly in the elderly. In regular full dosage NSAIDs have both a lasting analgesic and an anti-inflammatory effect making them useful for the management of continuous or regular pain associated with inflammation e.g. inflammatory arthritides. Pain relief starts soon after taking the first dose and a full analgesic effect should normally be obtained within a week, whereas an anti-inflammatory effect may not be achieved (or may not be clinically assessable) for up to 3 weeks.

Choice of NSAID

Ibuprofen has fewer side effects than other non-selective NSAIDs but its anti-inflammatory properties are weaker, making it unsuitable for conditions where inflammation is prominent e.g. acute gout.

Naproxen has good efficacy with a relatively low incidence of side effects (but more than ibuprofen); it is recommended first line treatment in acute gout.

Diclofenac is similar in efficacy to naproxen but is associated with a higher incidence of cardiovascular adverse events (and is in fact contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure).

Mefenamic acid has only minor anti-inflammatory properties and is typically used in the management of painful dysmenorrhoea. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment.

Risk of NSAID-induced GI adverse events	NSAIDs
Lowest risk	Ibuprofen
Intermediate risk	Naproxen, diclofenac, indometacin
Highest risk	Piroxicam, ketoprofen, ketorolac

Contraindications

NSAIDs are contraindicated in people with:

- A history of hypersensitivity or severe allergic reaction to aspirin or any other NSAID
- Severe heart failure (NSAIDs may impair renal function)
- Liver fibrosis, cirrhosis or acute liver failure (risk of variceal bleeding, hepatorenal syndrome and encephalopathy)
- Severe hepatic impairment (e.g. liver enzyme levels more than three times the upper limit of the normal range; serum albumin less than 25 g/L)
- Current treatment for gastrointestinal bleeding, symptomatic peptic ulcer, or gastrointestinal perforation or obstruction
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- Renal failure, with estimated glomerular filtration rate (eGFR) less than 30–15 mL/min/1.73 m2, or creatinine clearance less than 30–20 mL/min, or dehydration (relative)

Cautions

NSAIDs should be used with caution in:

- People with asthma (patients should be offered a trial to assess effect on asthma control)
- The elderly (increased risk of serious adverse effects such as gastrointestinal bleeding and perforation, which may be fatal)
- People with a history of peptic ulceration (standard NSAIDs contraindicated), or those at high risk of gastrointestinal adverse effects
- People with inflammatory bowel disease (NSAIDs may increase the risk of developing or cause exacerbations of ulcerative colitis or Crohn's disease)
- People with hepatitis or cholestasis (increased risk of gastrointestinal bleeding and fluid retention)
- Renal impairment (avoid if possible, sodium and water retention may occur leading to a deterioration in renal function and, possibly renal failure)
- People with heart failure (NSAIDs may impair renal function)
- People with hypertension (NSAIDs may impair renal function)
- In a women trying to conceive (NSAIDs may impair female fertility)

Adverse effects

Adverse effects may be minimised by selecting an appropriate NSAID and using the lowest effective dose for the shortest duration necessary to control symptoms.

Side effects of NSAIDs include:

- The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications such as ulcer, perforation, obstruction or bleeding (especially if risk factors are present – see below)
- Less commonly NSAIDs may cause cardiovascular and renal complications such as myocardial infarction, stroke, cardiac failure, hypertension, and renal failure (the risk is increased in people with IHD, CVD, PAD, CKD, HF or HTN, risk factors for cardiovascular disease and people > 65 years)
- Prolonged bleeding (for example after surgery) because of platelet inhibition
- Asthma (NSAIDs may exacerbate or precipitate asthma. Stop NSAID if it is suspected to have precipitated bronchospasm)
- Severe skin reactions and angioedema (stop NSAID)
- Very rarely, NSAIDs can precipitate severe hepatic reactions such as hepatitis, liver necrosis, or hepatic failure (stop NSAID)

Risk factors for increased risk of gastrointestinal adverse effects

People are at high risk of serious NSAID–induced gastrointestinal adverse events if they have one or more of the following risk factors:

- Using the maximum recommended dose of an NSAID
- Aged 65 years or older
- History of gastroduodenal ulcer, GI bleeding, or gastroduodenal perforation
- Concomitant use of medications that are known to increase the likelihood of upper GI adverse events (e.g. anticoagulants, aspirin, corticosteroids, and antidepressants (selective serotonin reuptake inhibitors, venlafaxine, or duloxetine))
- Serious comorbidity, such as cardiovascular disease, hepatic or renal impairment, diabetes, or hypertension
- Requirement for prolonged NSAID use including people with osteoarthritis or rheumatoid arthritis of any age and people aged 45 years or older with chronic low back pain
- The presence of Helicobacter pylori infection
- Excessive alcohol use
- Heavy smoking

Prevention and management of gastrointestinal adverse effects

- Preventing gastrointestinal adverse effects
  - Consider prescribing:
    - An alternative to an NSAID e.g. paracetamol +/- codeine
    - The lowest dose of NSAID for the shortest period of time
    - Only one NSAID at a time (avoid concomitant use with aspirin)
    - A COX-2 selective NSAID (but increased cardiovascular risk)
  - If the person is at an increased risk of GI adverse events and requires an NSAID, co-prescribe a proton pump inhibitor (PPI) for gastroprotection (e.g. omeprazole 20 mg od, lansoprazole 15 – 30 mg od)
- Managing gastrointestinal adverse effects
  - Where possible the NSAID should be withdrawn if an ulcer occurs
  - The ulcer should be treated with a PPI (a H2-receptor antagonist or misoprostol are alternatives)
  - On healing the patient should be tested for H. pylori and treated if required
  - If treatment with NSAID needs to continue, options include:
    - On healing continue PPI
    - Switch to selective COX-2 inhibitor

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Pharmacology: Gastro, Fluids and MSK

Question 5 of 49

Hartmann's solution contains how much bicarbonate:

a 111 mmol/L

b 5 mmol/L

c 11 mmol/L

d 50 mmol/L

e 29 mmol/L

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Pharmacology: Gastro, Fluids and MSK

Question 5 of 49

Hartmann's solution contains how much bicarbonate:

a) 111 mmol/L
b) **5 mmol/L** ✗
c) 11 mmol/L
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Answer

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
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Notes

Solutions of electrolytes are given intravenously to meet normal fluid and electrolyte requirements or to replenish substantial deficits or continuing losses when the patient is nauseated or vomiting and is unable to take adequate amounts by mouth. When intravenous administration is not possible, fluid (as sodium chloride 0.9% or glucose 5%) can also be given by subcutaneous infusion.

Sodium chloride

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Glucose solutions are also given in regimens with calcium and insulin for the emergency management of hyperkalaemia. They are also given after correction of hyperglycaemia during treatment of diabetic ketoacidosis, when they must be accompanied by continuing insulin infusion.

Potassium chloride mixtures

Potassium chloride with sodium chloride intravenous infusion is the initial treatment for the correction of severe hypokalaemia and when sufficient potassium cannot be taken by mouth. Repeated measurement of plasma-potassium concentration is necessary to avoid the development of hyperkalaemia, particularly in renal impairment. Initial potassium replacement therapy should not involve glucose infusions, because glucose may cause a further decrease in the plasma-potassium concentration.

Sodium bicarbonate

Sodium bicarbonate is used to control severe metabolic acidosis (pH < 7.1) particularly that caused by loss of bicarbonate (as in renal tubular acidosis or from excessive gastrointestinal losses). Mild metabolic acidosis associated with volume depletion should first be managed by appropriate fluid replacement because acidosis usually resolves as tissue and renal perfusion are restored.

In more severe metabolic acidosis or when the acidosis remains unresponsive to correction of anoxia or hypovolaemia, sodium bicarbonate (1.26%) can be infused over 3 – 4 hours with plasma-pH and electrolyte monitoring. In severe shock, for example in cardiac arrest, metabolic acidosis can develop without sodium or volume depletion; in these circumstances sodium bicarbonate is best given as a small volume of hypertonic solution, such as 50 mL of 8.4% solution intravenously.

Albumin

Albumin solutions, prepared from whole blood, contain soluble proteins and electrolytes but no clotting factors, blood group antibodies, or plasma cholinesterases thus may be given without regard to the recipient's blood group.

Albumin is usually used after the acute phase of illness to correct a plasma-volume deficit; hypoalbuminaemia itself is not an appropriate indication and the use of albumin solution in acute plasma or blood loss is wasteful where plasma substitutes should be used instead.

Concentrated albumin solution (20%) can be used under specialist supervision in patients with an intravascular fluid deficit and oedema because of interstitial fluid overload, to restore intravascular plasma volume with less exacerbation of the salt and water overload than isotonic solutions. Concentrated albumin solution may also be used to obtain a diuresis in hypoalbuminaemic patients (e.g. in hepatic cirrhosis).

Plasma substitutes

Dextran and gelatin may be used at the outset to expand and maintain blood volume in shock arising from conditions such as burns or septicaemia; they may also be used as an immediate short-term measure to treat haemorrhage until blood is available. Dextran and gelatin are rarely needed when shock is due to sodium and water depletion because, in these circumstances, the shock responds to water and electrolyte repletion.

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Pharmacology: Gastro, Fluids and MSK

Question 6 of 49

Regarding proton pump inhibitors, which of the following statements is CORRECT:

a They act by blocking the H^+/K^+ ATPase pump on gastric chief cells.

b They are protective against osteoporosis.

c They are associated with an increased risk of gastric malignancy.

d Post-endoscopic treatment of severe peptic ulcer bleeding typically includes a high dose oral PPI.

e They are associated with an increased risk of gastrointestinal infection.

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Pharmacology: Gastro, Fluids and MSK

Question 6 of 49

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b) They are protective against osteoporosis.
c) They are associated with an increased risk of gastric malignancy.
d) Post-endoscopic treatment of severe peptic ulcer bleeding typically includes a high dose oral PPI.
e) They are associated with an increased risk of gastrointestinal infection. ✔

Answer

PPIs are associated with an increased risk of gastrointestinal infection (including C. diff infection). PPIs act by inhibiting the proton pump on gastric parietal cells. Post-endoscopic treatment of severe peptic ulcer bleeding typically includes an intravenous high dose PPI which reduces risk of rebleeding and the need for surgery. PPIs are not associated with an increased risk of gastric malignancy, but may mask symptoms of malignancy. Long term PPI use is associated with an increased risk of fractures thus is not protective against osteoporosis.

Notes

Mechanism of action

Proton pump inhibitors act by directly inhibiting the H^+/K^+ ATPase (proton) pump on gastric parietal cell luminal membranes and thus inhibiting acid secretion.

Indications

Proton pump inhibitors may be indicated for:

- treatment of gastric and duodenal ulcers
- eradication of Helicobacter pylori in combination with antibacterials
- treatment of dyspepsia and GORD
- post-endoscopic treatment of severe peptic ulcer bleeding (an intravenous high dose PPI reduces risk of rebleeding and the need for surgery)
- treatment and prevention of NSAID-associated ulcers
- cystic fibrosis (reduces degradation of pancreatic enzyme supplements)
- Zollinger-Ellison syndrome (controls excessive gastric acid secretion)

Cautions

Proton pump inhibitors:

- can increase the risk of fractures (particularly when used at high doses for over a year in the elderly or used in those at risk of osteoporosis)
- may increase the risk of gastrointestinal infections (including Clostridium difficile infection)
- may mask the symptoms of gastric cancer (in patients with 'alarm features', gastric malignancy should be ruled out before starting treatment)
- can cause false negative H. pylori test results (and should be stopped at least 2 weeks before testing)

Side effects

Common side effects (usually mild and reversible) include:

- abdominal pain
- constipation
- diarrhoea
- flatulence
- headache
- nausea
- vomiting
- dizziness
- skin rashes

Possible adverse effects of long-term PPI treatment:

- Hypomagnesaemia (measurement of serum magnesium concentrations should be considered before and during prolonged treatment with a PPI, especially when used with other drugs that cause hypomagnesaemia or with digoxin)
- Rebound acid hypersecretion and protracted dyspepsia after stopping prolonged treatment
- Interstitial nephritis
- Vitamin B12 deficiency

Administration

Omeprazole:

- H. pylori eradication regimen dose: 20 mg od
- Healing peptic ulcer: 20 mg od (40 mg od may be used in severe or recurrent cases)
- Healing NSAID-associated ulcer: 20 mg od
- Prophylaxis for NSAID-associated ulcer: 20 mg od
- Major peptic ulcer bleeding (following endoscopic treatment): Initially 80 mg, to be given over 40–60 minutes, then (by continuous intravenous infusion) 8 mg/hour for 72 hours, subsequent dose then changed to oral therapy

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Pharmacology: Gastro, Fluids and MSK

Question 7 of 49

Which of the following treatments for hyperkalaemia actually removes potassium from the body:

a Salbutamol

b Insulin

c Sodium bicarbonate

d Calcium resonium

e Calcium gluconate

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Pharmacology: Gastro, Fluids and MSK

Question 7 of 49

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a) Salbutamol ✗
b) Insulin
c) Sodium bicarbonate
d) Calcium resonium ✓
e) Calcium gluconate

Answer

Calcium resonium is an ion-exchange resin that exchanges sodium for potassium as it passes through the intestine, leading to excretion of potassium from the body. Salbutamol and insulin act to increase intracellular uptake of K+ via Na-K ATP pump. Sodium bicarbonate acts to correct acidosis and thus promotes intracellular uptake of K+. Calcium gluconate acts to protect the cardiac membrane and has no effect on serum K+ levels.

Notes

Acute severe hyperkalaemia is characterised by a plasma-potassium concentration above 6.5 mmol/litre or the presence of ECG changes. ECG changes observed in hyperkalaemia may include tall peaked T waves, flattening or loss of P waves, broadening of QRS complexes, PR prolongation and bradycardia.

Emergency management

1. Reduce cardiac cell membrane excitability:
   - **10 ml calcium gluconate 10% by slow intravenous injection over 5 minutes**
     - ECG changes should improve within 1 – 3 minutes, but only has a transient effect of about 30 minutes
     - If there is no improvement, a further 10 ml should be given every 5 – 10 minutes until the ECG normalises (max 3 doses in total)
     - If the patient is taking digoxin, the calcium gluconate should be given slowly (mixed with 100 ml 5% glucose and given over 20 minutes) as rapid calcium administration may precipitate myocardial digoxin toxicity
2. Transcellular shift from extracellular to intracellular space:
   - **Intravenous infusion of insulin-glucose given as 10 units of Actrapid added to 50 ml of glucose 50% infused over 30 minutes**
     - Potassium should decrease by about 0.6 – 1.0 mmol/L in 15 minutes and the reduction lasts for about 60 minutes
     - This is repeated if necessary or a continuous infusion initiated
   - **Nebulised salbutamol 10 mg**
     - Reduces potassium by about 0.5 – 1.0 mmol/L in 15 – 30 minutes and lasts for about two hours
     - Should be used with caution in patients with ischaemic heart disease and history of cardiac arrhythmias
     - Response is attenuated in patients on beta-blockers and digoxin
   - The correction of causal or compounding acidosis with sodium bicarbonate infusion should be considered (if pH < 7.2)
     - Correction of acidosis promotes intracellular uptake of K+
3. Reduce total body potassium:
   - Any potassium supplements should be stopped, as well as any drugs that might precipitate hyperkalaemia (e.g. digoxin, beta-blockers)
   - Consider low potassium diet
   - Urinary potassium loss should be promoted with the use of appropriate fluids or diuretics to maintain good urine output
   - **Calcium resonium 15 g PO TDS**
     - Ion-exchange resin that exchanges sodium for potassium as it passes through the intestine
     - Should be given with regular laxatives to prevent constipation
     - Each gram removes approximately 1 mmol/L of potassium but onset is slow, taking 2 – 6 hours or longer
     - Contraindicated in patients with pre-existing hypercalcaemia

If the patient does not respond to the above measures dialysis will be required. DIalysis is likely to be needed if potassium is very high (> 7.5 mmol/L), if the patient is oligo/anuric, if the patient is already on long term dialysis or if the patient has advanced CKD.

Treatment	Administration	Mechanism of Action	Time to Onset of Action	Duration of Action	Achievable reduction of serum K+
Calcium resonium	15 g PO TDS	Ion-exchange resin that exchanges sodium for potassium as it passes through intestine	2 – 6 hours or longer	4 – 6 hours	Unknown
Calcium gluconate	10 ml calcium gluconate 10% by IV injection over 5 mins	Antagonises cardiac membrane excitability	1 – 3 minutes	30 minutes	N/A
Insulin-glucose infusion	10 units Actrapid in 50 ml glucose 50% infused over 30 mins	Increases intracellular uptake of K+ via Na-K ATP pump	Within 15 minutes	60 minutes	0.6 – 1 mmol/L
Nebulised salbutamol	10 mg nebulised	Increases intracellular uptake of K+ via Na-K ATP pump	Within 30 minutes	1 – 3 hours	0.5 – 1 mmol/L
Sodium bicarbonate	1.4% 500 ml IV over 2 hours	Corrects acidosis and thus promotes intracellular uptake of K+	After 60 minutes	Unknown	Unknown

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Pharmacology: Gastro, Fluids and MSK

Question 8 of 49

Hartmann's solution contains which of the following:

a Sodium and chloride

b Sodium, chloride and bicarbonate

c Sodium, chloride and potassium

d Sodium, chloride, bicarbonate, potassium and calcium

e Sodium, chloride, bicarbonate and potassium

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Pharmacology: Gastro, Fluids and MSK

Question 8 of 49

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a) **Sodium and chloride** ✖
b) Sodium, chloride and bicarbonate
c) Sodium, chloride and potassium
d) Sodium, chloride, bicarbonate, potassium and calcium ✔
e) Sodium, chloride, bicarbonate and potassium

Answer

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
- HCO_3^- 29 mmol/L
- Cl^- 111 mmol/L
- Ca^{2+} 2 mmol/L

Notes

Solutions of electrolytes are given intravenously to meet normal fluid and electrolyte requirements or to replenish substantial deficits or continuing losses when the patient is nauseated or vomiting and is unable to take adequate amounts by mouth. When intravenous administration is not possible, fluid (as sodium chloride 0.9% or glucose 5%) can also be given by subcutaneous infusion.

Sodium chloride

Normal saline (sodium chloride 0.9%) contains:

- Na^+ 150 mmol/L
- Cl^- 150 mmol/L

Sodium chloride in isotonic solution (0.9%) provides the most important extracellular ions in near physiological concentrations and is indicated in sodium depletion which can arise from such conditions as gastroenteritis, diabetic ketoacidosis, ileus, and ascites.

Chronic hyponatraemia arising from inappropriate secretion of antidiuretic hormone should ideally be corrected by fluid restriction. If sodium chloride is required for acute or chronic hyponatraemia, regardless of the cause, the deficit should be corrected slowly to avoid the risk of osmotic demyelination syndrome and the rise in plasma sodium concentration should not exceed 10 mmol/L in 24 hours. In severe hyponatraemia, sodium chloride 1.8 % may be used cautiously.

Hartmann's solution

Hartmann's solution (compound sodium lactate) can be used instead of isotonic sodium chloride solution during or after surgery, or in the initial management of the injured or wounded; it may reduce the risk of hyperchloraemic acidosis.

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
- HCO_3^- 29 mmol/L
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Glucose

Glucose solutions (5%) are used mainly to replace water deficit. Average water requirements in a healthy adult are 1.5 to 2.5 litres daily and this is needed to balance unavoidable losses of water through the skin and lungs and to provide sufficient for urinary excretion. Dehydration may occur when these losses are not compensated for by intake e.g. in coma, or in the elderly. Excessive loss of water without loss of electrolyte is uncommon, occurring in fevers, hyperthyroidism, diabetes insipidus and hypercalcaemia.

Glucose solutions are also used to correct and prevent hypoglycaemia and to provide a source of energy in those too ill to be fed adequately by mouth.

Glucose solutions are also given in regimens with calcium and insulin for the emergency management of hyperkalaemia. They are also given after correction of hyperglycaemia during treatment of diabetic ketoacidosis, when they must be accompanied by continuing insulin infusion.

Potassium chloride mixtures

Potassium chloride with sodium chloride intravenous infusion is the initial treatment for the correction of severe hypokalaemia and when sufficient potassium cannot be taken by mouth. Repeated measurement of plasma-potassium concentration is necessary to avoid the development of hyperkalaemia, particularly in renal impairment. Initial potassium replacement therapy should not involve glucose infusions, because glucose may cause a further decrease in the plasma-potassium concentration.

Sodium bicarbonate

Sodium bicarbonate is used to control severe metabolic acidosis (pH < 7.1) particularly that caused by loss of bicarbonate (as in renal tubular acidosis or from excessive gastrointestinal losses). Mild metabolic acidosis associated with volume depletion should first be managed by appropriate fluid replacement because acidosis usually resolves as tissue and renal perfusion are restored.

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Albumin solutions, prepared from whole blood, contain soluble proteins and electrolytes but no clotting factors, blood group antibodies, or plasma cholinesterases thus may be given without regard to the recipient's blood group.

Albumin is usually used after the acute phase of illness to correct a plasma-volume deficit; hypoalbuminaemia itself is not an appropriate indication and the use of albumin solution in acute plasma or blood loss is wasteful where plasma substitutes should be used instead.

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Plasma substitutes

Dextran and gelatin may be used at the outset to expand and maintain blood volume in shock arising from conditions such as burns or septicaemia; they may also be used as an immediate short-term measure to treat haemorrhage until blood is available. Dextran and gelatin are rarely needed when shock is due to sodium and water depletion because, in these circumstances, the shock responds to water and electrolyte repletion.

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Pharmacology: Gastro, Fluids and MSK

Question 9 of 49

Proton pump inhibitors act on which of the following cell types:

a Gastric parietal cells

b Gastric chief cells

c Gastric enteroendocrine cells

d G-cells

e Gastric acinar cells

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Pharmacology: Gastro, Fluids and MSK



Question 9 of 49

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Notes

Mechanism of action

Proton pump inhibitors act by directly inhibiting the H^+/K^+ ATPase (proton) pump on gastric parietal cell luminal membranes and thus inhibiting acid secretion.

Indications

Proton pump inhibitors may be indicated for:

- treatment of gastric and duodenal ulcers
- eradication of Helicobacter pylori in combination with antibacterials
- treatment of dyspepsia and GORD
- post-endoscopic treatment of severe peptic ulcer bleeding (an intravenous high dose PPI reduces risk of rebleeding and the need for surgery)
- treatment and prevention of NSAID-associated ulcers
- cystic fibrosis (reduces degradation of pancreatic enzyme supplements)
- Zollinger-Ellison syndrome (controls excessive gastric acid secretion)

Cautions

Proton pump inhibitors:

- can increase the risk of fractures (particularly when used at high doses for over a year in the elderly or used in those at risk of osteoporosis)
- may increase the risk of gastrointestinal infections (including Clostridium difficile infection)
- may mask the symptoms of gastric cancer (in patients with 'alarm features', gastric malignancy should be ruled out before starting treatment)
- can cause false negative H. pylori test results (and should be stopped at least 2 weeks before testing)

Side effects

Common side effects (usually mild and reversible) include:

- abdominal pain
- constipation
- diarrhoea
- flatulence
- headache
- nausea
- vomiting
- dizziness
- skin rashes

Possible adverse effects of long-term PPI treatment:

- Hypomagnesaemia (measurement of serum magnesium concentrations should be considered before and during prolonged treatment with a PPI, especially when used with other drugs that cause hypomagnesaemia or with digoxin)
- Rebound acid hypersecretion and protracted dyspepsia after stopping prolonged treatment
- Interstitial nephritis
- Vitamin B12 deficiency

Administration

Omeprazole:

- H. pylori eradication regimen dose: 20 mg od
- Healing peptic ulcer: 20 mg od (40 mg od may be used in severe or recurrent cases)
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Pharmacology: Gastro, Fluids and MSK

Question 10 of 49

Regarding potassium replacement, which of the following statements is INCORRECT:

a Hypokalaemia predisposes to arrhythmias particularly in patients taking digoxin.

b Compensation for potassium loss is particularly necessary in patients in whom secondary hyperaldosteronism occurs.

c Oral potassium supplements are often used for patients on loop or thiazide diuretics.

d Nausea and vomiting are common side effects of oral potassium salts and often limit compliance.

e Hypokalaemia is frequently associated with chloride depletion and metabolic alkalosis and this should also be corrected.

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Pharmacology: Gastro, Fluids and MSK

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c) Oral potassium supplements are often used for patients on loop or thiazide diuretics.

d) Nausea and vomiting are common side effects of oral potassium salts and often limit compliance.

e)
Hypokalaemia is frequently associated with chloride depletion and metabolic alkalosis and this should also be corrected.

Answer

Potassium supplements are seldom required with the small doses of diuretics given to treat hypertension; potassium-sparing diuretics (rather than potassium supplements) are recommended for prevention of hypokalaemia due to diuretics such as furosemide or the thiazides when these are given to eliminate oedema.

Notes

Compensation for potassium loss is particularly necessary:

- in those taking digoxin or antiarrhythmic drugs, where potassium depletion may induce arrhythmias
- in patients in whom secondary hyperaldosteronism occurs, e.g. renal artery stenosis, cirrhosis of the liver, nephrotic syndrome, and severe heart failure
- in patients with excessive losses of potassium in the faeces, e.g. chronic diarrhoea associated with intestinal malabsorption or laxative abuse

Other groups which may be prone to hypokalaemia include:

- the elderly who frequently take inadequate amounts of potassium in the diet (although this must be balanced with the risks of renal insufficiency)
- patients on long-term drug therapy with drugs known to cause hypokalaemia e.g. corticosteroids, or diuretics

Potassium salts may be given by mouth to prevent deficiencies or to treat established deficiencies of mild or moderate degree. Potassium salts cause nausea/vomiting and poor compliance is a major limitation to their effectiveness.

In established hypokalaemia, larger oral or intravenous doses may be required; hypokalaemia is frequently associated with chloride depletion and metabolic alkalosis and this must also be corrected.

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Pharmacology: Gastro, Fluids and MSK

Question 11 of 49

Regarding NSAIDs, which of the following statements is INCORRECT:

a Of the non-selective NSAIDs, diclofenac is associated with the greatest risk of thrombotic events.

b Selective COX-2 inhibitors have no gastrointestinal effects.

c NSAIDs may cause bronchospasm in patients with asthma.

d NSAIDs may cause prolonged bleeding after surgery because of platelet inhibition.

e The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications.

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Pharmacology: Gastro, Fluids and MSK

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b) Selective COX-2 inhibitors have no gastrointestinal effects.
c) NSAIDs may cause bronchospasm in patients with asthma.
d) NSAIDs may cause prolonged bleeding after surgery because of platelet inhibition.
e)
The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications.

Answer

All NSAIDs are associated with serious gastrointestinal toxicity. Evidence on the relative safety of non-selective NSAIDs indicates differences in the risks of serious upper gastrointestinal side effects:

- piroxicam, ketoprofen, and ketorolac trometamol are associated with the highest risk;
- indometacin, diclofenac, and naproxen are associated with intermediate risk,
- and ibuprofen with the lowest risk (although high doses of ibuprofen have been associated with intermediate risk).

Selective inhibitors of cyclo-oxygenase-2 are associated with a lower risk of serious upper gastrointestinal side-effects than non-selective NSAIDs.

All NSAID use (including cyclo-oxygenase-2 selective inhibitors) can, to varying degrees, be associated with a small increased risk of thrombotic events; cyclo-oxygenase-2 selective inhibitors, diclofenac and high-dose ibuprofen are associated with the greatest risk of thrombotic events.

Notes

Mechanism of action

NSAIDs reduce the production of prostaglandins by inhibiting the cyclo-oxygenase (COX) enzymes with analgesic, antipyretic and anti-inflammatory effects. The two main types of COX enzyme are COX-1 (which produces prostaglandins that help to maintain gastric mucosal integrity and platelet-initiated blood clotting) and COX-2 (which produces prostaglandins that mediate pain and inflammation). NSAIDs vary in their selectivity for inhibiting different types of COX; specific inhibition of COX-2 (e.g. celecoxib and etoricoxib) is associated with less gastrointestinal intolerance.

Therapeutic effects

In single doses NSAIDs have analgesic activity comparable to that of paracetamol, therefore given their side effect profile, paracetamol is preferred, particularly in the elderly. In regular full dosage NSAIDs have both a lasting analgesic and an anti-inflammatory effect making them useful for the management of continuous or regular pain associated with inflammation e.g. inflammatory arthritides. Pain relief starts soon after taking the first dose and a full analgesic effect should normally be obtained within a week, whereas an anti-inflammatory effect may not be achieved (or may not be clinically assessable) for up to 3 weeks.

Choice of NSAID

Ibuprofen has fewer side effects than other non-selective NSAIDs but its anti-inflammatory properties are weaker, making it unsuitable for conditions where inflammation is prominent e.g. acute gout.

Naproxen has good efficacy with a relatively low incidence of side effects (but more than ibuprofen); it is recommended first line treatment in acute gout.

Diclofenac is similar in efficacy to naproxen but is associated with a higher incidence of cardiovascular adverse events (and is in fact contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure).

Mefenamic acid has only minor anti-inflammatory properties and is typically used in the management of painful dysmenorrhoea. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment.

Risk of NSAID-induced GI adverse events	NSAIDs
Lowest risk	Ibuprofen
Intermediate risk	Naproxen, diclofenac, indometacin
Highest risk	Piroxicam, ketoprofen, ketorolac

Contraindications

NSAIDs are contraindicated in people with:

- A history of hypersensitivity or severe allergic reaction to aspirin or any other NSAID
- Severe heart failure (NSAIDs may impair renal function)
- Liver fibrosis, cirrhosis or acute liver failure (risk of variceal bleeding, hepatorenal syndrome and encephalopathy)
- Severe hepatic impairment (e.g. liver enzyme levels more than three times the upper limit of the normal range; serum albumin less than 25 g/L)
- Current treatment for gastrointestinal bleeding, symptomatic peptic ulcer, or gastrointestinal perforation or obstruction
- Coxibs and diclofenac are also contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, mild, moderate, or severe heart failure
- Renal failure, with estimated glomerular filtration rate (eGFR) less than 30–15 mL/min/1.73 m2, or creatinine clearance less than 30–20 mL/min, or dehydration (relative)

Cautions

NSAIDs should be used with caution in:

- People with asthma (patients should be offered a trial to assess effect on asthma control)
- The elderly (increased risk of serious adverse effects such as gastrointestinal bleeding and perforation, which may be fatal)
- People with a history of peptic ulceration (standard NSAIDs contraindicated), or those at high risk of gastrointestinal adverse effects
- People with inflammatory bowel disease (NSAIDs may increase the risk of developing or cause exacerbations of ulcerative colitis or Crohn's disease)
- People with hepatitis or cholestasis (increased risk of gastrointestinal bleeding and fluid retention)
- Renal impairment (avoid if possible, sodium and water retention may occur leading to a deterioration in renal function and, possibly renal failure)
- People with heart failure (NSAIDs may impair renal function)
- People with hypertension (NSAIDs may impair renal function)
- In a women trying to conceive (NSAIDs may impair female fertility)

Adverse effects

Adverse effects may be minimised by selecting an appropriate NSAID and using the lowest effective dose for the shortest duration necessary to control symptoms.

Side effects of NSAIDs include:

- The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications such as ulcer, perforation, obstruction or bleeding (especially if risk factors are present – see below)
- Less commonly NSAIDs may cause cardiovascular and renal complications such as myocardial infarction, stroke, cardiac failure, hypertension, and renal failure (the risk is increased in people with IHD, CVD, PAD, CKD, HF or HTN, risk factors for cardiovascular disease and people > 65 years)
- Prolonged bleeding (for example after surgery) because of platelet inhibition
- Asthma (NSAIDs may exacerbate or precipitate asthma. Stop NSAID if it is suspected to have precipitated bronchospasm)
- Severe skin reactions and angioedema (stop NSAID)
- Very rarely, NSAIDs can precipitate severe hepatic reactions such as hepatitis, liver necrosis, or hepatic failure (stop NSAID)

Risk factors for increased risk of gastrointestinal adverse effects

People are at high risk of serious NSAID–induced gastrointestinal adverse events if they have one or more of the following risk factors:

- Using the maximum recommended dose of an NSAID
- Aged 65 years or older
- History of gastroduodenal ulcer, GI bleeding, or gastroduodenal perforation
- Concomitant use of medications that are known to increase the likelihood of upper GI adverse events (e.g. anticoagulants, aspirin, corticosteroids, and antidepressants (selective serotonin reuptake inhibitors, venlafaxine, or duloxetine))
- Serious comorbidity, such as cardiovascular disease, hepatic or renal impairment, diabetes, or hypertension
- Requirement for prolonged NSAID use including people with osteoarthritis or rheumatoid arthritis of any age and people aged 45 years or older with chronic low back pain
- The presence of Helicobacter pylori infection
- Excessive alcohol use
- Heavy smoking

Prevention and management of gastrointestinal adverse effects

- Preventing gastrointestinal adverse effects
  - Consider prescribing:
    - An alternative to an NSAID e.g. paracetamol +/- codeine
    - The lowest dose of NSAID for the shortest period of time
    - Only one NSAID at a time (avoid concomitant use with aspirin)
    - A COX-2 selective NSAID (but increased cardiovascular risk)
  - If the person is at an increased risk of GI adverse events and requires an NSAID, co-prescribe a proton pump inhibitor (PPI) for gastroprotection (e.g. omeprazole 20 mg od, lansoprazole 15 – 30 mg od)
- Managing gastrointestinal adverse effects
  - Where possible the NSAID should be withdrawn if an ulcer occurs
  - The ulcer should be treated with a PPI (a H2-receptor antagonist or misoprostol are alternatives)
  - On healing the patient should be tested for H. pylori and treated if required
  - If treatment with NSAID needs to continue, options include:
    - On healing continue PPI
    - Switch to selective COX-2 inhibitor

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Pharmacology: Gastro, Fluids and MSK

Question 12 of 49

Which of the following is NOT an adverse effect associated with NSAIDs:

a Renal impairment

b Bronchospasm in asthma

c Exacerbation of inflammatory bowel disease

d Increased risk of thrombotic events

e Increased risk of fracture

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Pharmacology: Gastro, Fluids and MSK

Question 12 of 49

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a) **Renal impairment** ✗
b) Bronchospasm in asthma
c) Exacerbation of inflammatory bowel disease
d) Increased risk of thrombotic events
e) Increased risk of fracture ✓

Answer

Increased risk of fracture is not an adverse feature of NSAIDs.

Side effects of NSAIDs include:

- The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications such as ulcer, perforation, obstruction or bleeding (especially if risk factors are present – see below)
- Less commonly NSAIDs may cause cardiovascular and renal complications such as myocardial infarction, stroke, cardiac failure, hypertension, and renal failure (the risk is increased in people with IHD, CVD, PAD, CKD, HF or HTN, risk factors for cardiovascular disease and people > 65 years)
- Prolonged bleeding (for example after surgery) because of platelet inhibition
- Asthma (NSAIDs may exacerbate or precipitate asthma. Stop NSAID if it is suspected to have precipitated bronchospasm)
- Severe skin reactions and angioedema (stop NSAID)
- Very rarely, NSAIDs can precipitate severe hepatic reactions such as hepatitis, liver necrosis, or hepatic failure (stop NSAID)

Notes

Mechanism of action

NSAIDs reduce the production of prostaglandins by inhibiting the cyclo-oxygenase (COX) enzymes with analgesic, antipyretic and anti-inflammatory effects. The two main types of COX enzyme are COX-1 (which produces prostaglandins that help to maintain gastric mucosal integrity and platelet-initiated blood clotting) and COX-2 (which produces prostaglandins that mediate pain and inflammation). NSAIDs vary in their selectivity for inhibiting different types of COX; specific inhibition of COX-2 (e.g. celecoxib and etoricoxib) is associated with less gastrointestinal intolerance.

Therapeutic effects

In single doses NSAIDs have analgesic activity comparable to that of paracetamol, therefore given their side effect profile, paracetamol is preferred, particularly in the elderly. In regular full dosage NSAIDs have both a lasting analgesic and an anti-inflammatory effect making them useful for the management of continuous or regular pain associated with inflammation e.g. inflammatory arthritides. Pain relief starts soon after taking the first dose and a full analgesic effect should normally be obtained within a week, whereas an anti-inflammatory effect may not be achieved (or may not be clinically assessable) for up to 3 weeks.

Choice of NSAID

Ibuprofen has fewer side effects than other non-selective NSAIDs but its anti-inflammatory properties are weaker, making it unsuitable for conditions where inflammation is prominent e.g. acute gout.

Naproxen has good efficacy with a relatively low incidence of side effects (but more than ibuprofen); it is recommended first line treatment in acute gout.

Diclofenac is similar in efficacy to naproxen but is associated with a higher incidence of cardiovascular adverse events (and is in fact contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure).

Mefenamic acid has only minor anti-inflammatory properties and is typically used in the management of painful dysmenorrhoea. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment.

Risk of NSAID-induced GI adverse events	NSAIDs
Lowest risk	Ibuprofen
Intermediate risk	Naproxen, diclofenac, indometacin
Highest risk	Piroxicam, ketoprofen, ketorolac

Contraindications

NSAIDs are contraindicated in people with:

- A history of hypersensitivity or severe allergic reaction to aspirin or any other NSAID
- Severe heart failure (NSAIDs may impair renal function)
- Liver fibrosis, cirrhosis or acute liver failure (risk of variceal bleeding, hepatorenal syndrome and encephalopathy)
- Severe hepatic impairment (e.g. liver enzyme levels more than three times the upper limit of the normal range; serum albumin less than 25 g/L)
- Current treatment for gastrointestinal bleeding, symptomatic peptic ulcer, or gastrointestinal perforation or obstruction
- Coxibs and diclofenac are also contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, mild, moderate, or severe heart failure
- Renal failure, with estimated glomerular filtration rate (eGFR) less than 30–15 mL/min/1.73 m2, or creatinine clearance less than 30–20 mL/min, or dehydration (relative)

Cautions

NSAIDs should be used with caution in:

- People with asthma (patients should be offered a trial to assess effect on asthma control)
- The elderly (increased risk of serious adverse effects such as gastrointestinal bleeding and perforation, which may be fatal)
- People with a history of peptic ulceration (standard NSAIDs contraindicated), or those at high risk of gastrointestinal adverse effects
- People with inflammatory bowel disease (NSAIDs may increase the risk of developing or cause exacerbations of ulcerative colitis or Crohn's disease)
- People with hepatitis or cholestasis (increased risk of gastrointestinal bleeding and fluid retention)
- Renal impairment (avoid if possible, sodium and water retention may occur leading to a deterioration in renal function and, possibly renal failure)
- People with heart failure (NSAIDs may impair renal function)
- People with hypertension (NSAIDs may impair renal function)
- In a women trying to conceive (NSAIDs may impair female fertility)

Adverse effects

Adverse effects may be minimised by selecting an appropriate NSAID and using the lowest effective dose for the shortest duration necessary to control symptoms.

Side effects of NSAIDs include:

- The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications such as ulcer, perforation, obstruction or bleeding (especially if risk factors are present – see below)
- Less commonly NSAIDs may cause cardiovascular and renal complications such as myocardial infarction, stroke, cardiac failure, hypertension, and renal failure (the risk is increased in people with IHD, CVD, PAD, CKD, HF or HTN, risk factors for cardiovascular disease and people > 65 years)
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- Excessive alcohol use
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    - A COX-2 selective NSAID (but increased cardiovascular risk)
  - If the person is at an increased risk of GI adverse events and requires an NSAID, co-prescribe a proton pump inhibitor (PPI) for gastroprotection (e.g. omeprazole 20 mg od, lansoprazole 15 – 30 mg od)
- Managing gastrointestinal adverse effects
  - Where possible the NSAID should be withdrawn if an ulcer occurs
  - The ulcer should be treated with a PPI (a H2-receptor antagonist or misoprostol are alternatives)
  - On healing the patient should be tested for H. pylori and treated if required
  - If treatment with NSAID needs to continue, options include:
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    - Switch to selective COX-2 inhibitor

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Pharmacology: Gastro, Fluids and MSK

Question 13 of 49

Hartmann's solution contains how much calcium:

a 16 mmol/L

b 5 mmol/L

c 11 mmol/L

d 2 mmol/L

e 29 mmol/L

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Pharmacology: Gastro, Fluids and MSK

Question 13 of 49

Hartmann's solution contains how much calcium:

a) 16 mmol/L
b) 5 mmol/L
c) 11 mmol/L
d) **2 mmol/L** ✔
e) 29 mmol/L

Answer

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
- HCO_3^- 29 mmol/L
- Cl^- 111 mmol/L
- Ca^{2+} 2 mmol/L

Notes

Solutions of electrolytes are given intravenously to meet normal fluid and electrolyte requirements or to replenish substantial deficits or continuing losses when the patient is nauseated or vomiting and is unable to take adequate amounts by mouth. When intravenous administration is not possible, fluid (as sodium chloride 0.9% or glucose 5%) can also be given by subcutaneous infusion.

Sodium chloride

Normal saline (sodium chloride 0.9%) contains:

- Na^+ 150 mmol/L
- Cl^- 150 mmol/L

Sodium chloride in isotonic solution (0.9%) provides the most important extracellular ions in near physiological concentrations and is indicated in sodium depletion which can arise from such conditions as gastroenteritis, diabetic ketoacidosis, ileus, and ascites.

Chronic hyponatraemia arising from inappropriate secretion of antidiuretic hormone should ideally be corrected by fluid restriction. If sodium chloride is required for acute or chronic hyponatraemia, regardless of the cause, the deficit should be corrected slowly to avoid the risk of osmotic demyelination syndrome and the rise in plasma sodium concentration should not exceed 10 mmol/L in 24 hours. In severe hyponatraemia, sodium chloride 1.8 % may be used cautiously.

Hartmann's solution

Hartmann's solution (compound sodium lactate) can be used instead of isotonic sodium chloride solution during or after surgery, or in the initial management of the injured or wounded; it may reduce the risk of hyperchloraemic acidosis.

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
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- Cl^- 111 mmol/L
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Glucose

Glucose solutions (5%) are used mainly to replace water deficit. Average water requirements in a healthy adult are 1.5 to 2.5 litres daily and this is needed to balance unavoidable losses of water through the skin and lungs and to provide sufficient for urinary excretion. Dehydration may occur when these losses are not compensated for by intake e.g. in coma, or in the elderly. Excessive loss of water without loss of electrolyte is uncommon, occurring in fevers, hyperthyroidism, diabetes insipidus and hypercalcaemia.

Glucose solutions are also used to correct and prevent hypoglycaemia and to provide a source of energy in those too ill to be fed adequately by mouth.

Glucose solutions are also given in regimens with calcium and insulin for the emergency management of hyperkalaemia. They are also given after correction of hyperglycaemia during treatment of diabetic ketoacidosis, when they must be accompanied by continuing insulin infusion.

Potassium chloride mixtures

Potassium chloride with sodium chloride intravenous infusion is the initial treatment for the correction of severe hypokalaemia and when sufficient potassium cannot be taken by mouth. Repeated measurement of plasma-potassium concentration is necessary to avoid the development of hyperkalaemia, particularly in renal impairment. Initial potassium replacement therapy should not involve glucose infusions, because glucose may cause a further decrease in the plasma-potassium concentration.

Sodium bicarbonate

Sodium bicarbonate is used to control severe metabolic acidosis (pH < 7.1) particularly that caused by loss of bicarbonate (as in renal tubular acidosis or from excessive gastrointestinal losses). Mild metabolic acidosis associated with volume depletion should first be managed by appropriate fluid replacement because acidosis usually resolves as tissue and renal perfusion are restored.

In more severe metabolic acidosis or when the acidosis remains unresponsive to correction of anoxia or hypovolaemia, sodium bicarbonate (1.26%) can be infused over 3 – 4 hours with plasma-pH and electrolyte monitoring. In severe shock, for example in cardiac arrest, metabolic acidosis can develop without sodium or volume depletion; in these circumstances sodium bicarbonate is best given as a small volume of hypertonic solution, such as 50 mL of 8.4% solution intravenously.

Albumin

Albumin solutions, prepared from whole blood, contain soluble proteins and electrolytes but no clotting factors, blood group antibodies, or plasma cholinesterases thus may be given without regard to the recipient's blood group.

Albumin is usually used after the acute phase of illness to correct a plasma-volume deficit; hypoalbuminaemia itself is not an appropriate indication and the use of albumin solution in acute plasma or blood loss is wasteful where plasma substitutes should be used instead.

Concentrated albumin solution (20%) can be used under specialist supervision in patients with an intravascular fluid deficit and oedema because of interstitial fluid overload, to restore intravascular plasma volume with less exacerbation of the salt and water overload than isotonic solutions. Concentrated albumin solution may also be used to obtain a diuresis in hypoalbuminaemic patients (e.g. in hepatic cirrhosis).

Plasma substitutes

Dextran and gelatin may be used at the outset to expand and maintain blood volume in shock arising from conditions such as burns or septicaemia; they may also be used as an immediate short-term measure to treat haemorrhage until blood is available. Dextran and gelatin are rarely needed when shock is due to sodium and water depletion because, in these circumstances, the shock responds to water and electrolyte repletion.

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Pharmacology: Gastro, Fluids and MSK

Question 14 of 49

Hartmann's solution may be preferred to sodium chloride 0.9% as it may reduce the risk of:

a Hypernatraemia

b Hyponatraemia

c Fluid overload

d Hyperchloraemic acidosis

e Osmotic demyelination syndrome

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Pharmacology: Gastro, Fluids and MSK

Question 14 of 49

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b) Hyponatraemia
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Answer

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Pharmacology: Gastro, Fluids and MSK

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Hartmann's solution contains how much chloride:

a 131 mmol/L

b 150 mmol/L

c 111 mmol/L

d 94 mmol/L

e 126 mmol/L

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Pharmacology: Gastro, Fluids and MSK

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Pharmacology: Gastro, Fluids and MSK

Question 16 of 49

Regarding oral rehydration therapy, which of the following statements is INCORRECT:

a Diarrhoea is the most common indication for fluid and electrolyte replacement worldwide.

b Intestinal absorption of sodium and water is enhanced by glucose.

c Oral rehydration solutions should contain an alkalinising agent to counter acidosis.

d Oral rehydration solutions should be slightly hyperosmolar.

e Rehydration in hypernatraemic dehydration should occur more slowly.

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Pharmacology: Gastro, Fluids and MSK

Question 16 of 49

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c) Oral rehydration solutions should contain an alkalinising agent to counter acidosis.

d) Oral rehydration solutions should be slightly hyperosmolar.

e) Rehydration in hypernatraemic dehydration should occur more slowly.

Answer

Oral rehydration solutions should be slightly hypo-osmolar (about 250 mmol/litre) to prevent the possible induction of osmotic diarrhoea.

Notes

As a worldwide problem diarrhoea is by far the most important indication for fluid and electrolyte replacement.

Intestinal absorption of sodium and water is enhanced by glucose (and other carbohydrates). Replacement of fluid and electrolytes lost through diarrhoea can therefore be achieved by giving solutions (e.g. Dioralyte) containing sodium, potassium, and glucose or another carbohydrate such as rice starch.

Oral rehydration solutions should:

- enhance the absorption of water and electrolytes;
- replace the electrolyte deficit adequately and safely;
- contain an alkalinising agent to counter acidosis;
- be slightly hypo-osmolar (about 250 mmol/litre) to prevent the possible induction of osmotic diarrhoea;
- be simple to use in hospital and at home;
- be palatable and acceptable, especially to children;
- be readily available.

Rehydration should be rapid over 3 to 4 hours (except in hypernatraemic dehydration in which case rehydration should occur more slowly over 12 hours).

The patient should be reassessed after initial rehydration and if still dehydrated rapid fluid replacement should continue.

Once rehydration is complete further dehydration is prevented by encouraging the patient to drink normal volumes of an appropriate fluid and by replacing continuing losses with an oral rehydration solution.

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Pharmacology: Gastro, Fluids and MSK

Question 17 of 49

Which of the following best describes the mechanism by which nonsteroidal anti-inflammatory drugs (NSAIDs) have their therapeutic effect:

a Modulate serotonin release and uptake

b Decrease histamine release

c Decrease production of leukotrienes

d Decrease production of prostaglandins

e Decrease bradykinin release

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Pharmacology: Gastro, Fluids and MSK



Question 17 of 49

Which of the following best describes the mechanism by which nonsteroidal anti-inflammatory drugs (NSAIDs) have their therapeutic effect:

a) Modulate serotonin release and uptake ✗
b) Decrease histamine release
c) Decrease production of leukotrienes
d) Decrease production of prostaglandins ✓
e) Decrease bradykinin release

Answer

NSAIDs reduce the production of prostaglandins by inhibiting the cyclo-oxygenase (COX) enzymes with analgesic, antipyretic and anti-inflammatory effects.

Notes

Mechanism of action

NSAIDs reduce the production of prostaglandins by inhibiting the cyclo-oxygenase (COX) enzymes with analgesic, antipyretic and anti-inflammatory effects. The two main types of COX enzyme are COX-1 (which produces prostaglandins that help to maintain gastric mucosal integrity and platelet-initiated blood clotting) and COX-2 (which produces prostaglandins that mediate pain and inflammation). NSAIDs vary in their selectivity for inhibiting different types of COX; specific inhibition of COX-2 (e.g. celecoxib and etoricoxib) is associated with less gastrointestinal intolerance.

Therapeutic effects

In single doses NSAIDs have analgesic activity comparable to that of paracetamol, therefore given their side effect profile, paracetamol is preferred, particularly in the elderly. In regular full dosage NSAIDs have both a lasting analgesic and an anti-inflammatory effect making them useful for the management of continuous or regular pain associated with inflammation e.g. inflammatory arthritides. Pain relief starts soon after taking the first dose and a full analgesic effect should normally be obtained within a week, whereas an anti-inflammatory effect may not be achieved (or may not be clinically assessable) for up to 3 weeks.

Choice of NSAID

Ibuprofen has fewer side effects than other non-selective NSAIDs but its anti-inflammatory properties are weaker, making it unsuitable for conditions where inflammation is prominent e.g. acute gout.

Naproxen has good efficacy with a relatively low incidence of side effects (but more than ibuprofen); it is recommended first line treatment in acute gout.

Diclofenac is similar in efficacy to naproxen but is associated with a higher incidence of cardiovascular adverse events (and is in fact contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure).

Mefenamic acid has only minor anti-inflammatory properties and is typically used in the management of painful dysmenorrhoea. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment.

Risk of NSAID-induced GI adverse events	NSAIDs
Lowest risk	Ibuprofen
Intermediate risk	Naproxen, diclofenac, indometacin
Highest risk	Piroxicam, ketoprofen, ketorolac

Contraindications

NSAIDs are contraindicated in people with:

- A history of hypersensitivity or severe allergic reaction to aspirin or any other NSAID
- Severe heart failure (NSAIDs may impair renal function)
- Liver fibrosis, cirrhosis or acute liver failure (risk of variceal bleeding, hepatorenal syndrome and encephalopathy)
- Severe hepatic impairment (e.g. liver enzyme levels more than three times the upper limit of the normal range; serum albumin less than 25 g/L)
- Current treatment for gastrointestinal bleeding, symptomatic peptic ulcer, or gastrointestinal perforation or obstruction
- Coxibs and diclofenac are also contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, mild, moderate, or severe heart failure
- Renal failure, with estimated glomerular filtration rate (eGFR) less than 30–15 mL/min/1.73 m2, or creatinine clearance less than 30–20 mL/min, or dehydration (relative)

Cautions

NSAIDs should be used with caution in:

- People with asthma (patients should be offered a trial to assess effect on asthma control)
- The elderly (increased risk of serious adverse effects such as gastrointestinal bleeding and perforation, which may be fatal)
- People with a history of peptic ulceration (standard NSAIDs contraindicated), or those at high risk of gastrointestinal adverse effects
- People with inflammatory bowel disease (NSAIDs may increase the risk of developing or cause exacerbations of ulcerative colitis or Crohn's disease)
- People with hepatitis or cholestasis (increased risk of gastrointestinal bleeding and fluid retention)
- Renal impairment (avoid if possible, sodium and water retention may occur leading to a deterioration in renal function and, possibly renal failure)
- People with heart failure (NSAIDs may impair renal function)
- People with hypertension (NSAIDs may impair renal function)
- In a women trying to conceive (NSAIDs may impair female fertility)

Adverse effects

Adverse effects may be minimised by selecting an appropriate NSAID and using the lowest effective dose for the shortest duration necessary to control symptoms.

Side effects of NSAIDs include:

- The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications such as ulcer, perforation, obstruction or bleeding (especially if risk factors are present – see below)
- Less commonly NSAIDs may cause cardiovascular and renal complications such as myocardial infarction, stroke, cardiac failure, hypertension, and renal failure (the risk is increased in people with IHD, CVD, PAD, CKD, HF or HTN, risk factors for cardiovascular disease and people > 65 years)
- Prolonged bleeding (for example after surgery) because of platelet inhibition
- Asthma (NSAIDs may exacerbate or precipitate asthma. Stop NSAID if it is suspected to have precipitated bronchospasm)
- Severe skin reactions and angioedema (stop NSAID)
- Very rarely, NSAIDs can precipitate severe hepatic reactions such as hepatitis, liver necrosis, or hepatic failure (stop NSAID)

Risk factors for increased risk of gastrointestinal adverse effects

People are at high risk of serious NSAID–induced gastrointestinal adverse events if they have one or more of the following risk factors:

- Using the maximum recommended dose of an NSAID
- Aged 65 years or older
- History of gastroduodenal ulcer, GI bleeding, or gastroduodenal perforation
- Concomitant use of medications that are known to increase the likelihood of upper GI adverse events (e.g. anticoagulants, aspirin, corticosteroids, and antidepressants (selective serotonin reuptake inhibitors, venlafaxine, or duloxetine))
- Serious comorbidity, such as cardiovascular disease, hepatic or renal impairment, diabetes, or hypertension
- Requirement for prolonged NSAID use including people with osteoarthritis or rheumatoid arthritis of any age and people aged 45 years or older with chronic low back pain
- The presence of Helicobacter pylori infection
- Excessive alcohol use
- Heavy smoking

Prevention and management of gastrointestinal adverse effects

- Preventing gastrointestinal adverse effects
  - Consider prescribing:
    - An alternative to an NSAID e.g. paracetamol +/- codeine
    - The lowest dose of NSAID for the shortest period of time
    - Only one NSAID at a time (avoid concomitant use with aspirin)
    - A COX-2 selective NSAID (but increased cardiovascular risk)
  - If the person is at an increased risk of GI adverse events and requires an NSAID, co-prescribe a proton pump inhibitor (PPI) for gastroprotection (e.g. omeprazole 20 mg od, lansoprazole 15 – 30 mg od)
- Managing gastrointestinal adverse effects
  - Where possible the NSAID should be withdrawn if an ulcer occurs
  - The ulcer should be treated with a PPI (a H2-receptor antagonist or misoprostol are alternatives)
  - On healing the patient should be tested for H. pylori and treated if required
  - If treatment with NSAID needs to continue, options include:
    - On healing continue PPI
    - Switch to selective COX-2 inhibitor

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Pharmacology: Gastro, Fluids and MSK

Question 18 of 49

Regarding antacids, which of the following statements is CORRECT:

a Tablet preparations are more effective than liquid preparations.

b Magnesium carbonate tends to cause constipation.

c Antacids are contraindicated in hypophosphataemia.

d Antacids are best taken regularly each morning for maximum symptom control.

e Antacids increase absorption of some drugs.

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Pharmacology: Gastro, Fluids and MSK



Question 18 of 49

Regarding antacids, which of the following statements is CORRECT:

a) **Tablet preparations are more effective than liquid preparations.** ✗
b) Magnesium carbonate tends to cause constipation.
c) Antacids are contraindicated in hypophosphataemia. ✓
d) Antacids are best taken regularly each morning for maximum symptom control.
e) Antacids increase absorption of some drugs.

Answer

Antacids are contraindicated in hypophosphataemia. Liquid preparations are more effective than tablet preparations. Magnesium-containing antacids tend to be laxative whereas aluminium-containing antacids tend to be constipating. Antacids are best taken when symptoms occur or are expected, usually between meals and at bedtime. Antacids should preferably not be taken at the same time as other drugs since they may impair absorption.

Notes

Dyspepsia is upper abdominal discomfort or pain which may be described as a burning sensation, a heaviness, or an ache. It is often related to eating and may be accompanied by other symptoms such as nausea, fullness in the upper abdomen, or belching. The most common causes of dyspepsia are gastro-oesophageal reflux disease (GORD), peptic ulcer disease and non-ulcer (functional) dyspepsia.

Patients with dyspepsia/GORD should be advised that symptoms may improve if they:

- lose weight (if they are overweight)
- stop smoking
- reduce alcohol consumption
- reduce intake of any food or drink associated with worsening symptoms
- avoid having meals within 3 – 4 hours of going to bed
- raise the height of the head of the bed by a few inches

Antacids

Antacids are used for symptomatic relief in dyspepsia and in GORD.

Mechanism of action

Antacids work by neutralising gastric acid and raising the luminal pH. However, the effect is only for a relatively short period, requiring frequent administration. Antacids do promote ulcer healing, but less well than anti-secretory drugs.

Administration

Antacids are best taken when symptoms occur or are expected, usually between meals and at bedtime. Liquid preparations are more effective than tablet preparations.

Interactions

Antacids should preferably not be taken at the same time as other drugs since they may impair absorption. Antacids may damage enteric coatings designed to prevent dissolution in the stomach.

Side effects

Aluminium- and magnesium-containing antacids (e.g. aluminium hydroxide, magnesium carbonate), being relatively insoluble in water, are long acting if retained in the stomach. Magnesium-containing antacids tend to be laxative whereas aluminium-containing antacids tend to be constipating; antacids containing both magnesium and aluminium may reduce these colonic side effects.

Contraindications

Antacids are contraindicated in hypophosphataemia.

Alginates

Alginates taken in combination with an antacid increases the viscosity of stomach contents and can protect the oesophageal mucosa from acid reflux. Some alginate-containing preparations e.g. Gaviscon form a viscous gel raft that floats on the surface of the stomach contents, thereby reducing symptoms of reflux.

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Pharmacology: Gastro, Fluids and MSK

Question 19 of 49

Regarding the management of gout, which of the following statements is CORRECT:

a Allopurinol is effective at treating an acute attack of gout.

b Hyperuricaemia is a common side effect of allopurinol.

c Side effects are uncommon with colchicine.

d Paracetamol is indicated first line for acute gout.

e Oral corticosteroids may be considered if first line treatments aren't effective in treating acute gout.

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Pharmacology: Gastro, Fluids and MSK

Question 19 of 49

Regarding the management of gout, which of the following statements is CORRECT:

a) Allopurinol is effective at treating an acute attack of gout. ✗

b) Hyperuricaemia is a common side effect of allopurinol.

c) Side effects are uncommon with colchicine.

d) Paracetamol is indicated first line for acute gout.

e) ✓

Oral corticosteroids may be considered if first line treatments aren't effective in treating acute gout.

Answer

Oral or parenteral corticosteroids are an effective alternative in those who cannot tolerate or who are resistant to NSAIDs and colchicine. Paracetamol may be used as an adjunctive therapy, but is not indicated on its own as it has no anti-inflammatory properties. Side effects are common with colchicine therapy (typically nausea, vomiting, diarrhoea and abdominal pain) and may be severe enough to limit therapy. Allopurinol is not used for acute gout and if started during an acute attack may prolong the attack indefinitely. Allopurinol reduces uric acid formation from purines and may be used to prevent further attacks of gout by correcting hyperuricaemia.

Notes

Gout is a disorder of purine metabolism characterised by a raised uric acid level in the blood (hyperuricaemia) and the deposition of urate crystals in joints and other tissues, such as soft connective tissues or the urinary tract.

NSAIDs

Acute attacks of gout are usually treated with an NSAID such as diclofenac, indometacin, or naproxen. Treatment should be started as soon as possible and continued until 48 hours after the attack has resolved. A PPI should be co-prescribed for gastric protection in people at high risk of gastrointestinal adverse events.

Colchicine

Colchicine is an alternative in patients in whom NSAIDs are contraindicated, not tolerated or ineffective. Although its use is limited by toxicity at higher doses, it is useful in patients with heart failure or those taking anticoagulants.

The dose is 500 micrograms 2 – 4 times a day until the symptoms are relieved or diarrhoea or vomiting occurs (max 6 mg per course and do not repeat treatment within 3 days).

Colchicine should be avoided in people with blood dyscrasias and bone marrow disease.

Common side effects of colchicine include:

- abdominal pain
- nausea and vomiting
- diarrhoea

Side effects are dose dependent and may be severe enough to limit treatment.

Corticosteroids

Oral or parenteral corticosteroids are an effective alternative in those who cannot tolerate or who are resistant to NSAIDs and colchicine. Intra-articular injection of a corticosteroid can be used occasionally in acute monoarticular gout.

Allopurinol

Allopurinol is not effective in treating an acute attack and may prolong it indefinitely if started during the acute episode, but should be continued during an acute attack if the patient is already established on long term therapy.

Long term treatment (prevention) of gout may be indicated in patients with frequent recurrence of acute attacks of gout, the presence of tophi, or signs of chronic gouty arthritis. Allopurinol reduces uric acid formation from purines and may be used to prevent further attacks of gout by correcting hyperuricaemia. The initiation of treatment may precipitate an acute attack, and should be covered with an NSAID or colchicine, continued for at least one month after the hyperuricaemia has been corrected.

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Pharmacology: Gastro, Fluids and MSK

Question 20 of 49

You have diagnosed acute flare up of gout in a 55 year old man. You would like to start him on an NSAID. However his wife was recently diagnosed with a peptic ulcer on endoscopy secondary to NSAID use. Which of the following NSAIDs is least likely to cause GI side effects:

a Naproxen

b Ibuprofen

c Diclofenac

d Indometacin

e Piroxicam

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Pharmacology: Gastro, Fluids and MSK



Question 20 of 49

You have diagnosed acute flare up of gout in a 55 year old man. You would like to start him on an NSAID. However his wife was recently diagnosed with a peptic ulcer on endoscopy secondary to NSAID use. Which of the following NSAIDs is least likely to cause GI side effects:

a) Naproxen ✘
b) Ibuprofen ✔
c) Diclofenac
d) Indometacin
e) Piroxicam

Answer

All NSAIDs are associated with serious gastrointestinal toxicity. Evidence on the relative safety of non-selective NSAIDs indicates differences in the risks of serious upper gastrointestinal side effects:

- piroxicam, ketoprofen, and ketorolac trometamol are associated with the highest risk;
- indometacin, diclofenac, and naproxen are associated with intermediate risk,
- and ibuprofen with the lowest risk (although high doses of ibuprofen have been associated with intermediate risk).

Selective inhibitors of cyclo-oxygenase-2 are associated with a lower risk of serious upper gastrointestinal side-effects than non-selective NSAIDs.

Notes

Mechanism of action

NSAIDs reduce the production of prostaglandins by inhibiting the cyclo-oxygenase (COX) enzymes with analgesic, antipyretic and anti-inflammatory effects. The two main types of COX enzyme are COX-1 (which produces prostaglandins that help to maintain gastric mucosal integrity and platelet-initiated blood clotting) and COX-2 (which produces prostaglandins that mediate pain and inflammation). NSAIDs vary in their selectivity for inhibiting different types of COX; specific inhibition of COX-2 (e.g. celecoxib and etoricoxib) is associated with less gastrointestinal intolerance.

Therapeutic effects

In single doses NSAIDs have analgesic activity comparable to that of paracetamol, therefore given their side effect profile, paracetamol is preferred, particularly in the elderly. In regular full dosage NSAIDs have both a lasting analgesic and an anti-inflammatory effect making them useful for the management of continuous or regular pain associated with inflammation e.g. inflammatory arthritides. Pain relief starts soon after taking the first dose and a full analgesic effect should normally be obtained within a week, whereas an anti-inflammatory effect may not be achieved (or may not be clinically assessable) for up to 3 weeks.

Choice of NSAID

Ibuprofen has fewer side effects than other non-selective NSAIDs but its anti-inflammatory properties are weaker, making it unsuitable for conditions where inflammation is prominent e.g. acute gout.

Naproxen has good efficacy with a relatively low incidence of side effects (but more than ibuprofen); it is recommended first line treatment in acute gout.

Diclofenac is similar in efficacy to naproxen but is associated with a higher incidence of cardiovascular adverse events (and is in fact contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure).

Mefenamic acid has only minor anti-inflammatory properties and is typically used in the management of painful dysmenorrhoea. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment.

Risk of NSAID-induced GI adverse events	NSAIDs
Lowest risk	Ibuprofen
Intermediate risk	Naproxen, diclofenac, indometacin
Highest risk	Piroxicam, ketoprofen, ketorolac

Contraindications

NSAIDs are contraindicated in people with:

- A history of hypersensitivity or severe allergic reaction to aspirin or any other NSAID
- Severe heart failure (NSAIDs may impair renal function)
- Liver fibrosis, cirrhosis or acute liver failure (risk of variceal bleeding, hepatorenal syndrome and encephalopathy)
- Severe hepatic impairment (e.g. liver enzyme levels more than three times the upper limit of the normal range; serum albumin less than 25 g/L)
- Current treatment for gastrointestinal bleeding, symptomatic peptic ulcer, or gastrointestinal perforation or obstruction
- Coxibs and diclofenac are also contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, mild, moderate, or severe heart failure
- Renal failure, with estimated glomerular filtration rate (eGFR) less than 30–15 mL/min/1.73 m2, or creatinine clearance less than 30–20 mL/min, or dehydration (relative)

Cautions

NSAIDs should be used with caution in:

- People with asthma (patients should be offered a trial to assess effect on asthma control)
- The elderly (increased risk of serious adverse effects such as gastrointestinal bleeding and perforation, which may be fatal)
- People with a history of peptic ulceration (standard NSAIDs contraindicated), or those at high risk of gastrointestinal adverse effects
- People with inflammatory bowel disease (NSAIDs may increase the risk of developing or cause exacerbations of ulcerative colitis or Crohn's disease)
- People with hepatitis or cholestasis (increased risk of gastrointestinal bleeding and fluid retention)
- Renal impairment (avoid if possible, sodium and water retention may occur leading to a deterioration in renal function and, possibly renal failure)
- People with heart failure (NSAIDs may impair renal function)
- People with hypertension (NSAIDs may impair renal function)
- In a women trying to conceive (NSAIDs may impair female fertility)

Adverse effects

Adverse effects may be minimised by selecting an appropriate NSAID and using the lowest effective dose for the shortest duration necessary to control symptoms.

Side effects of NSAIDs include:

- The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications such as ulcer, perforation, obstruction or bleeding (especially if risk factors are present – see below)
- Less commonly NSAIDs may cause cardiovascular and renal complications such as myocardial infarction, stroke, cardiac failure, hypertension, and renal failure (the risk is increased in people with IHD, CVD, PAD, CKD, HF or HTN, risk factors for cardiovascular disease and people > 65 years)
- Prolonged bleeding (for example after surgery) because of platelet inhibition
- Asthma (NSAIDs may exacerbate or precipitate asthma. Stop NSAID if it is suspected to have precipitated bronchospasm)
- Severe skin reactions and angioedema (stop NSAID)
- Very rarely, NSAIDs can precipitate severe hepatic reactions such as hepatitis, liver necrosis, or hepatic failure (stop NSAID)

Risk factors for increased risk of gastrointestinal adverse effects

People are at high risk of serious NSAID–induced gastrointestinal adverse events if they have one or more of the following risk factors:

- Using the maximum recommended dose of an NSAID
- Aged 65 years or older
- History of gastroduodenal ulcer, GI bleeding, or gastroduodenal perforation
- Concomitant use of medications that are known to increase the likelihood of upper GI adverse events (e.g. anticoagulants, aspirin, corticosteroids, and antidepressants (selective serotonin reuptake inhibitors, venlafaxine, or duloxetine))
- Serious comorbidity, such as cardiovascular disease, hepatic or renal impairment, diabetes, or hypertension
- Requirement for prolonged NSAID use including people with osteoarthritis or rheumatoid arthritis of any age and people aged 45 years or older with chronic low back pain
- The presence of Helicobacter pylori infection
- Excessive alcohol use
- Heavy smoking

Prevention and management of gastrointestinal adverse effects

- Preventing gastrointestinal adverse effects
  - Consider prescribing:
    - An alternative to an NSAID e.g. paracetamol +/- codeine
    - The lowest dose of NSAID for the shortest period of time
    - Only one NSAID at a time (avoid concomitant use with aspirin)
    - A COX-2 selective NSAID (but increased cardiovascular risk)
  - If the person is at an increased risk of GI adverse events and requires an NSAID, co-prescribe a proton pump inhibitor (PPI) for gastroprotection (e.g. omeprazole 20 mg od, lansoprazole 15 – 30 mg od)
- Managing gastrointestinal adverse effects
  - Where possible the NSAID should be withdrawn if an ulcer occurs
  - The ulcer should be treated with a PPI (a H2-receptor antagonist or misoprostol are alternatives)
  - On healing the patient should be tested for H. pylori and treated if required
  - If treatment with NSAID needs to continue, options include:
    - On healing continue PPI
    - Switch to selective COX-2 inhibitor

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Pharmacology: Gastro, Fluids and MSK

Question 21 of 49

Intravenous glucose solutions are typically used in the treatment of all but which of the following:

a Diabetic ketoacidosis

b Hypoglycaemia

c Hypokalaemia

d Routine fluid maintenance in patients who are nil by mouth

e Hyperkalaemia

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Pharmacology: Gastro, Fluids and MSK

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Intravenous glucose solutions are typically used in the treatment of all but which of the following:

a) **Diabetic ketoacidosis** ✗
b) Hypoglycaemia
c) Hypokalaemia ✓
d) Routine fluid maintenance in patients who are nil by mouth
e) Hyperkalaemia

Answer

Initial potassium replacement therapy in hypokalaemia should not involve glucose infusions, because glucose may cause a further decrease in the plasma-potassium concentration. Glucose infusions are used for the other indications listed above.

Notes

Solutions of electrolytes are given intravenously to meet normal fluid and electrolyte requirements or to replenish substantial deficits or continuing losses when the patient is nauseated or vomiting and is unable to take adequate amounts by mouth. When intravenous administration is not possible, fluid (as sodium chloride 0.9% or glucose 5%) can also be given by subcutaneous infusion.

Sodium chloride

Normal saline (sodium chloride 0.9%) contains:

- Na^+ 150 mmol/L
- Cl^- 150 mmol/L

Sodium chloride in isotonic solution (0.9%) provides the most important extracellular ions in near physiological concentrations and is indicated in sodium depletion which can arise from such conditions as gastroenteritis, diabetic ketoacidosis, ileus, and ascites.

Chronic hyponatraemia arising from inappropriate secretion of antidiuretic hormone should ideally be corrected by fluid restriction. If sodium chloride is required for acute or chronic hyponatraemia, regardless of the cause, the deficit should be corrected slowly to avoid the risk of osmotic demyelination syndrome and the rise in plasma sodium concentration should not exceed 10 mmol/L in 24 hours. In severe hyponatraemia, sodium chloride 1.8 % may be used cautiously.

Hartmann's solution

Hartmann's solution (compound sodium lactate) can be used instead of isotonic sodium chloride solution during or after surgery, or in the initial management of the injured or wounded; it may reduce the risk of hyperchloraemic acidosis.

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
- HCO_3^- 29 mmol/L
- Cl^- 111 mmol/L
- Ca^{2+} 2 mmol/L

Glucose

Glucose solutions (5%) are used mainly to replace water deficit. Average water requirements in a healthy adult are 1.5 to 2.5 litres daily and this is needed to balance unavoidable losses of water through the skin and lungs and to provide sufficient for urinary excretion. Dehydration may occur when these losses are not compensated for by intake e.g. in coma, or in the elderly. Excessive loss of water without loss of electrolyte is uncommon, occurring in fevers, hyperthyroidism, diabetes insipidus and hypercalcaemia.

Glucose solutions are also used to correct and prevent hypoglycaemia and to provide a source of energy in those too ill to be fed adequately by mouth.

Glucose solutions are also given in regimens with calcium and insulin for the emergency management of hyperkalaemia. They are also given after correction of hyperglycaemia during treatment of diabetic ketoacidosis, when they must be accompanied by continuing insulin infusion.

Potassium chloride mixtures

Potassium chloride with sodium chloride intravenous infusion is the initial treatment for the correction of severe hypokalaemia and when sufficient potassium cannot be taken by mouth. Repeated measurement of plasma-potassium concentration is necessary to avoid the development of hyperkalaemia, particularly in renal impairment. Initial potassium replacement therapy should not involve glucose infusions, because glucose may cause a further decrease in the plasma-potassium concentration.

Sodium bicarbonate

Sodium bicarbonate is used to control severe metabolic acidosis (pH < 7.1) particularly that caused by loss of bicarbonate (as in renal tubular acidosis or from excessive gastrointestinal losses). Mild metabolic acidosis associated with volume depletion should first be managed by appropriate fluid replacement because acidosis usually resolves as tissue and renal perfusion are restored.

In more severe metabolic acidosis or when the acidosis remains unresponsive to correction of anoxia or hypovolaemia, sodium bicarbonate (1.26%) can be infused over 3 – 4 hours with plasma-pH and electrolyte monitoring. In severe shock, for example in cardiac arrest, metabolic acidosis can develop without sodium or volume depletion; in these circumstances sodium bicarbonate is best given as a small volume of hypertonic solution, such as 50 mL of 8.4% solution intravenously.

Albumin

Albumin solutions, prepared from whole blood, contain soluble proteins and electrolytes but no clotting factors, blood group antibodies, or plasma cholinesterases thus may be given without regard to the recipient's blood group.

Albumin is usually used after the acute phase of illness to correct a plasma-volume deficit; hypoalbuminaemia itself is not an appropriate indication and the use of albumin solution in acute plasma or blood loss is wasteful where plasma substitutes should be used instead.

Concentrated albumin solution (20%) can be used under specialist supervision in patients with an intravascular fluid deficit and oedema because of interstitial fluid overload, to restore intravascular plasma volume with less exacerbation of the salt and water overload than isotonic solutions. Concentrated albumin solution may also be used to obtain a diuresis in hypoalbuminaemic patients (e.g. in hepatic cirrhosis).

Plasma substitutes

Dextran and gelatin may be used at the outset to expand and maintain blood volume in shock arising from conditions such as burns or septicaemia; they may also be used as an immediate short-term measure to treat haemorrhage until blood is available. Dextran and gelatin are rarely needed when shock is due to sodium and water depletion because, in these circumstances, the shock responds to water and electrolyte repletion.

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Pharmacology: Gastro, Fluids and MSK

Question 22 of 49

Regarding antimotility drugs, which of the following statements is INCORRECT:

a Loperamide acts on opioid receptors in the gastrointestinal tract.

b They are useful for symptomatic relief of acute diarrhoea in children.

c They are useful for symptomatic relief of acute diarrhoea in adults.

d They are contraindicated in active ulcerative colitis.

e They are contraindicated in antibiotic-associated colitis.

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Pharmacology: Gastro, Fluids and MSK

Question 22 of 49

Regarding antimotility drugs, which of the following statements is INCORRECT:

a) **Loperamide acts on opioid receptors in the gastrointestinal tract.** ✗
b) They are useful for symptomatic relief of acute diarrhoea in children. ✓
c) They are useful for symptomatic relief of acute diarrhoea in adults.
d) They are contraindicated in active ulcerative colitis.
e) They are contraindicated in antibiotic-associated colitis.

Answer

They may be used in the management of uncomplicated acute diarrhoea in adults but are not recommended for acute diarrhoea in young children.

Notes

Acute diarrhoea

The priority in the management of acute diarrhoea, such as in gastroenteritis, is the prevention or reversal of fluid and electrolyte depletion. This is particularly important in infants, and in frail and elderly patients. Oral rehydration preparations may be used, or in more severe cases patients may be admitted to hospital and given intravenous fluid and electrolyte replacement.

Antimotility drugs

Antimotility drugs can relieve symptoms of acute diarrhoea by decreasing intestinal secretion and motility, prolonging intestinal transit time and facilitating water reabsorption (e.g. loperamide acting on opioid mu-receptors).

They may be used in the management of uncomplicated acute diarrhoea in adults but are not recommended for acute diarrhoea in young children.

Antimotility drugs are contraindicated in active ulcerative colitis, antibiotic-associated colitis and conditions where abdominal distension develops or where inhibition of peristalsis should be avoided.

Antispasmodics

Antispasmodics are occasionally of value in treating abdominal cramp associated with diarrhoea in adults but they should not be used for primary treatment.

Antispasmodics and antiemetics should be avoided in young children with gastroenteritis because they are rarely effective and have troublesome side effects.

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Pharmacology: Gastro, Fluids and MSK

Question 23 of 49

Regarding the management of gout, which of the following statements is CORRECT:

a Colchicine is first line for management of acute gout.

b Colchicine is used as a long term therapy for prevention of gout.

c Allopurinol may cause an acute attack of gout when first started.

d Colchicine is contraindicated in patients taking anticoagulants.

e Ibuprofen is the NSAID of choice in treating acute gout.

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Pharmacology: Gastro, Fluids and MSK



Question 23 of 49

Regarding the management of gout, which of the following statements is CORRECT:

a) **Colchicine is first line for management of acute gout.** ✗
b) Colchicine is used as a long term therapy for prevention of gout.
c) Allopurinol may cause an acute attack of gout when first started. ✓
d) Colchicine is contraindicated in patients taking anticoagulants.
e) Ibuprofen is the NSAID of choice in treating acute gout.

Answer

Allopurinol is a treatment for the prevention of gout. The initiation of treatment may precipitate an acute attack, and should be covered with an NSAID or colchicine, continued for at least one month after the hyperuricaemia has been corrected. Colchicine is not indicated for long term prevention of gout. NSAIDs are first line for acute gout; colchicine is an alternative in whom NSAIDs are contraindicated, not tolerated or ineffective (including in people taking anticoagulants). Ibuprofen is not recommended for acute gout, given it has only weak anti-inflammatory properties; naproxen, diclofenac or indometacin are indicated instead.

Notes

Gout is a disorder of purine metabolism characterised by a raised uric acid level in the blood (hyperuricaemia) and the deposition of urate crystals in joints and other tissues, such as soft connective tissues or the urinary tract.

NSAIDs

Acute attacks of gout are usually treated with an NSAID such as diclofenac, indometacin, or naproxen. Treatment should be started as soon as possible and continued until 48 hours after the attack has resolved. A PPI should be co-prescribed for gastric protection in people at high risk of gastrointestinal adverse events.

Colchicine

Colchicine is an alternative in patients in whom NSAIDs are contraindicated, not tolerated or ineffective. Although its use is limited by toxicity at higher doses, it is useful in patients with heart failure or those taking anticoagulants.

The dose is 500 micrograms 2 – 4 times a day until the symptoms are relieved or diarrhoea or vomiting occurs (max 6 mg per course and do not repeat treatment within 3 days).

Colchicine should be avoided in people with blood dyscrasias and bone marrow disease.

Common side effects of colchicine include:

- abdominal pain
- nausea and vomiting
- diarrhoea

Side effects are dose dependent and may be severe enough to limit treatment.

Corticosteroids

Oral or parenteral corticosteroids are an effective alternative in those who cannot tolerate or who are resistant to NSAIDs and colchicine. Intra-articular injection of a corticosteroid can be used occasionally in acute monoarticular gout.

Allopurinol

Allopurinol is not effective in treating an acute attack and may prolong it indefinitely if started during the acute episode, but should be continued during an acute attack if the patient is already established on long term therapy.

Long term treatment (prevention) of gout may be indicated in patients with frequent recurrence of acute attacks of gout, the presence of tophi, or signs of chronic gouty arthritis. Allopurinol reduces uric acid formation from purines and may be used to prevent further attacks of gout by correcting hyperuricaemia. The initiation of treatment may precipitate an acute attack, and should be covered with an NSAID or colchicine, continued for at least one month after the hyperuricaemia has been corrected.

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Pharmacology: Gastro, Fluids and MSK

Question 24 of 49

A 31 year old female patient presents to ED complaining of some symptoms she suspects are due to a drug that she has recently started. Her GP has started her on hyoscine butylbromide for symptomatic relief of irritable bowel syndrome. Which of the following would you least expect to see as a side effect in this patient:

a Blurred vision

b Diarrhoea

c Tachycardia

d Dry mouth

e Urinary retention

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Question 24 of 49

A 31 year old female patient presents to ED complaining of some symptoms she suspects are due to a drug that she has recently started. Her GP has started her on hyoscine butylbromide for symptomatic relief of irritable bowel syndrome. Which of the following would you least expect to see as a side effect in this patient:

a) **Blurred vision** ✗
b) Diarrhoea ✓
c) Tachycardia
d) Dry mouth
e) Urinary retention

Answer

Hyoscine butylbromide is an antimuscarinic drug which is used as an antispasmodic. Typical side effects of antimuscarinics include:

- dilation of pupils with loss of accommodation (cycloplegia) and photophobia resulting in blurred vision ("Blind as a bat")
- dry mouth, eyes and skin ("Dry as a bone")
- elevated temperature ("Hot as a hare")
- skin flushing ("Red as a beet")
- confusion or agitation particularly in the elderly ("Mad as a hatter")
- reduced bronchial secretions
- transient bradycardia followed by tachycardia, palpitation and arrhythmias
- urinary retention
- constipation

Notes

Antimuscarinics

Antimuscarinics reduce intestinal motility (by acting on acetylcholine receptors and relaxing smooth muscle) and can be used as antispasmodics for the management of irritable bowel syndrome. Hyoscine butylbromide is advocated as a gastrointestinal antispasmodic, but it is poorly absorbed and thus has limited clinical utility.

Other indications for antimuscarinic drugs include arrhythmias, asthma and airways disease, motion sickness, parkinsonism, urinary incontinence, mydriasis and cycloplegia, premedication, and as an antidote to organophosphate poisoning.

Cautions

Antimuscarinics should be used with caution in:

- acute myocardial infarction
- arrhythmias (may be worsened)
- autonomic neuropathy
- cardiac insufficiency (due to association with tachycardia)
- cardiac surgery (due to association with tachycardia)
- children (increased risk of side effects)
- conditions characterised by tachycardia
- congestive heart failure (may be worsened)
- coronary artery disease (may be worsened)
- diarrhoea
- elderly (especially if frail)
- gastro-oesophageal reflux disease
- hiatus hernia with reflux oesophagitis
- hypertension
- hyperthyroidism (due to association with tachycardia)
- individuals susceptible to angle-closure glaucoma
- prostatic hyperplasia
- pyrexia
- ulcerative colitis

Contraindications

Antimuscarinics are contraindicated in:

- gastrointestinal obstruction
- intestinal atony
- myasthenia gravis
- paralytic ileus
- prostatic enlargement
- pyloric stenosis
- severe ulcerative colitis
- significant bladder outflow obstruction
- toxic megacolon
- urinary retention

Side effects

Many drugs have antimuscarinic effects; concomitant use of two or more such drugs can increase side effects such as dry mouth, urine retention, and constipation.

Side effects of antimuscarinics include:

- dilation of pupils with loss of accommodation (cycloplegia) and photophobia resulting in blurred vision ("Blind as a bat")
- dry mouth, eyes and skin ("Dry as a bone")
- elevated temperature ("Hot as a hare")
- skin flushing ("Red as a beet")
- confusion or agitation particularly in the elderly ("Mad as a hatter")
- reduced bronchial secretions
- transient bradycardia followed by tachycardia, palpitation and arrhythmias
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Pharmacology: Gastro, Fluids and MSK

Question 25 of 49

Which of the following lifestyle changes is not likely to improve symptoms of dyspepsia/gastro-oesophageal reflux disease (GORD):

a Lowering the head of the bed

b Avoiding alcohol

c Smoking cessation

d Weight loss

e Cutting our spicy food

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Question 25 of 49

Which of the following lifestyle changes is not likely to improve symptoms of dyspepsia/gastro-oesophageal reflux disease (GORD):

a) **Lowering the head of the bed** ✔
b) Avoiding alcohol
c) Smoking cessation
d) Weight loss
e) Cutting our spicy food

Answer

Patients with dyspepsia/GORD should be advised that symptoms may improve if they:

- lose weight (if they are overweight)
- stop smoking
- reduce alcohol consumption
- reduce intake of any food or drink associated with worsening symptoms
- avoid having meals within 3 – 4 hours of going to bed
- raise the height of the head of the bed by a few inches

Notes

Dyspepsia is upper abdominal discomfort or pain which may be described as a burning sensation, a heaviness, or an ache. It is often related to eating and may be accompanied by other symptoms such as nausea, fullness in the upper abdomen, or belching. The most common causes of dyspepsia are gastro-oesophageal reflux disease (GORD), peptic ulcer disease and non-ulcer (functional) dyspepsia.

Patients with dyspepsia/GORD should be advised that symptoms may improve if they:

- lose weight (if they are overweight)
- stop smoking
- reduce alcohol consumption
- reduce intake of any food or drink associated with worsening symptoms
- avoid having meals within 3 – 4 hours of going to bed
- raise the height of the head of the bed by a few inches

Antacids

Antacids are used for symptomatic relief in dyspepsia and in GORD.

Mechanism of action

Antacids work by neutralising gastric acid and raising the luminal pH. However, the effect is only for a relatively short period, requiring frequent administration. Antacids do promote ulcer healing, but less well than anti-secretory drugs.

Administration

Antacids are best taken when symptoms occur or are expected, usually between meals and at bedtime. Liquid preparations are more effective than tablet preparations.

Interactions

Antacids should preferably not be taken at the same time as other drugs since they may impair absorption. Antacids may damage enteric coatings designed to prevent dissolution in the stomach.

Side effects

Aluminium- and magnesium-containing antacids (e.g. aluminium hydroxide, magnesium carbonate), being relatively insoluble in water, are long acting if retained in the stomach. Magnesium-containing antacids tend to be laxative whereas aluminium-containing antacids tend to be constipating; antacids containing both magnesium and aluminium may reduce these colonic side effects.

Contraindications

Antacids are contraindicated in hypophosphataemia.

Alginates

Alginates taken in combination with an antacid increases the viscosity of stomach contents and can protect the oesophageal mucosa from acid reflux. Some alginate-containing preparations e.g. Gaviscon form a viscous gel raft that floats on the surface of the stomach contents, thereby reducing symptoms of reflux.

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Pharmacology: Gastro, Fluids and MSK

Question 26 of 49

Regarding NSAIDs, which of the following statements is CORRECT:

a Selective inhibition of COX-1 is associated with less gastrointestinal intolerance.

b The anti-inflammatory effect is usually apparent after 24 hours.

c In single doses, NSAIDs have far greater analgesic properties than paracetamol.

d Mefenamic acid has very potent anti-inflammatory properties.

e Diclofenac is contraindicated in people with ischaemic heart disease.

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Pharmacology: Gastro, Fluids and MSK



Question 26 of 49

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a) Selective inhibition of COX-1 is associated with less gastrointestinal intolerance. ✖

b) The anti-inflammatory effect is usually apparent after 24 hours.

c) In single doses, NSAIDs have far greater analgesic properties than paracetamol.

d) Mefenamic acid has very potent anti-inflammatory properties.

e) Diclofenac is contraindicated in people with ischaemic heart disease. ✔

Answer

Due to their increased risk of cardiovascular adverse events, coxibs and diclofenac are contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure. Other NSAIDs are only contraindicated in people with severe heart failure. Selective inhibition of COX-2 is associated with less gastrointestinal intolerance. In single doses NSAIDs have analgesic activity comparable to that of paracetamol, therefore given their side effect profile, paracetamol is preferred, particularly in the elderly. Pain relief starts soon after taking the first dose and a full analgesic effect should normally be obtained within a week, whereas an anti-inflammatory effect may not be achieved (or may not be clinically assessable) for up to 3 weeks. Mefenamic acid has only very mild anti-inflammatory properties.

Notes

Mechanism of action

NSAIDs reduce the production of prostaglandins by inhibiting the cyclo-oxygenase (COX) enzymes with analgesic, antipyretic and anti-inflammatory effects. The two main types of COX enzyme are COX-1 (which produces prostaglandins that help to maintain gastric mucosal integrity and platelet-initiated blood clotting) and COX-2 (which produces prostaglandins that mediate pain and inflammation). NSAIDs vary in their selectivity for inhibiting different types of COX; specific inhibition of COX-2 (e.g. celecoxib and etoricoxib) is associated with less gastrointestinal intolerance.

Therapeutic effects

In single doses NSAIDs have analgesic activity comparable to that of paracetamol, therefore given their side effect profile, paracetamol is preferred, particularly in the elderly. In regular full dosage NSAIDs have been a lasting analgesic and an anti-inflammatory effect making them useful for the management of continuous or regular pain associated with inflammation e.g. inflammatory arthritides. Pain relief starts soon after taking the first dose and a full analgesic effect should normally be obtained within a week, whereas an anti-inflammatory effect may not be achieved (or may not be clinically assessable) for up to 3 weeks.

Choice of NSAID

Ibuprofen has fewer side effects than other non-selective NSAIDs but its anti-inflammatory properties are weaker, making it unsuitable for conditions where inflammation is prominent e.g. acute gout.

Naproxen has good efficacy with a relatively low incidence of side effects (but more than ibuprofen); it is recommended first line treatment in acute gout.

Diclofenac is similar in efficacy to naproxen but is associated with a higher incidence of cardiovascular adverse events (and is in fact contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure).

Mefenamic acid has only minor anti-inflammatory properties and is typically used in the management of painful dysmenorrhoea. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment.

Risk of NSAID-induced GI adverse events	NSAIDs
Lowest risk	Ibuprofen
Intermediate risk	Naproxen, diclofenac, indometacin
Highest risk	Piroxicam, ketoprofen, ketorolac

Contraindications

NSAIDs are contraindicated in people with:

- A history of hypersensitivity or severe allergic reaction to aspirin or any other NSAID
- Severe heart failure (NSAIDs may impair renal function)
- Liver fibrosis, cirrhosis or acute liver failure (risk of variceal bleeding, hepatorenal syndrome and encephalopathy)
- Severe hepatic impairment (e.g. liver enzyme levels more than three times the upper limit of the normal range; serum albumin less than 25 g/L)
- Current treatment for gastrointestinal bleeding, symptomatic peptic ulcer, or gastrointestinal perforation or obstruction
- Coxibs and diclofenac are also contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, mild, moderate, or severe heart failure
- Renal failure, with estimated glomerular filtration rate (eGFR) less than 30–15 mL/min/1.73 m2, or creatinine clearance less than 30–20 mL/min, or dehydration (relative)

Cautions

NSAIDs should be used with caution in:

- People with asthma (patients should be offered a trial to assess effect on asthma control)
- The elderly (increased risk of serious adverse effects such as gastrointestinal bleeding and perforation, which may be fatal)
- People with a history of peptic ulceration (standard NSAIDs contraindicated), or those at high risk of gastrointestinal adverse effects
- People with inflammatory bowel disease (NSAIDs may increase the risk of developing or cause exacerbations of ulcerative colitis or Crohn's disease)
- People with hepatitis or cholestasis (increased risk of gastrointestinal bleeding and fluid retention)
- Renal impairment (avoid if possible, sodium and water retention may occur leading to a deterioration in renal function and, possibly renal failure)
- People with heart failure (NSAIDs may impair renal function)
- People with hypertension (NSAIDs may impair renal function)
- In a women trying to conceive (NSAIDs may impair female fertility)

Adverse effects

Adverse effects may be minimised by selecting an appropriate NSAID and using the lowest effective dose for the shortest duration necessary to control symptoms.

Side effects of NSAIDs include:

- The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications such as ulcer, perforation, obstruction or bleeding (especially if risk factors are present – see below)
- Less commonly NSAIDs may cause cardiovascular and renal complications such as myocardial infarction, stroke, cardiac failure, hypertension, and renal failure (the risk is increased in people with IHD, CVD, PAD, CKD, HF or HTN, risk factors for cardiovascular disease and people > 65 years)
- Prolonged bleeding (for example after surgery) because of platelet inhibition
- Asthma (NSAIDs may exacerbate or precipitate asthma. Stop NSAID if it is suspected to have precipitated bronchospasm)
- Severe skin reactions and angioedema (stop NSAID)
- Very rarely, NSAIDs can precipitate severe hepatic reactions such as hepatitis, liver necrosis, or hepatic failure (stop NSAID)

Risk factors for increased risk of gastrointestinal adverse effects

People are at high risk of serious NSAID–induced gastrointestinal adverse events if they have one or more of the following risk factors:

- Using the maximum recommended dose of an NSAID
- Aged 65 years or older
- History of gastroduodenal ulcer, GI bleeding, or gastroduodenal perforation
- Concomitant use of medications that are known to increase the likelihood of upper GI adverse events (e.g. anticoagulants, aspirin, corticosteroids, and antidepressants (selective serotonin reuptake inhibitors, venlafaxine, or duloxetine))
- Serious comorbidity, such as cardiovascular disease, hepatic or renal impairment, diabetes, or hypertension
- Requirement for prolonged NSAID use including people with osteoarthritis or rheumatoid arthritis of any age and people aged 45 years or older with chronic low back pain
- The presence of Helicobacter pylori infection
- Excessive alcohol use
- Heavy smoking

Prevention and management of gastrointestinal adverse effects

- Preventing gastrointestinal adverse effects
  - Consider prescribing:
    - An alternative to an NSAID e.g. paracetamol +/- codeine
    - The lowest dose of NSAID for the shortest period of time
    - Only one NSAID at a time (avoid concomitant use with aspirin)
    - A COX-2 selective NSAID (but increased cardiovascular risk)
  - If the person is at an increased risk of GI adverse events and requires an NSAID, co-prescribe a proton pump inhibitor (PPI) for gastroprotection (e.g. omeprazole 20 mg od, lansoprazole 15 – 30 mg od)
- Managing gastrointestinal adverse effects
  - Where possible the NSAID should be withdrawn if an ulcer occurs
  - The ulcer should be treated with a PPI (a H2-receptor antagonist or misoprostol are alternatives)
  - On healing the patient should be tested for H. pylori and treated if required
  - If treatment with NSAID needs to continue, options include:
    - On healing continue PPI
    - Switch to selective COX-2 inhibitor

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Pharmacology: Gastro, Fluids and MSK

Question 27 of 49

Hartmann's solution contains how much sodium:

a 131 mmol/L

b 150 mmol/L

c 111 mmol/L

d 94 mmol/L

e 126 mmol/L

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Question 27 of 49

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a) 131 mmol/L ✔
b) 150 mmol/L
c) 111 mmol/L
d) 94 mmol/L
e) 126 mmol/L

Answer

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
- HCO_3^- 29 mmol/L
- Cl^- 111 mmol/L
- Ca^{2+} 2 mmol/L

Notes

Solutions of electrolytes are given intravenously to meet normal fluid and electrolyte requirements or to replenish substantial deficits or continuing losses when the patient is nauseated or vomiting and is unable to take adequate amounts by mouth. When intravenous administration is not possible, fluid (as sodium chloride 0.9% or glucose 5%) can also be given by subcutaneous infusion.

Sodium chloride

Normal saline (sodium chloride 0.9%) contains:

- Na^+ 150 mmol/L
- Cl^- 150 mmol/L

Sodium chloride in isotonic solution (0.9%) provides the most important extracellular ions in near physiological concentrations and is indicated in sodium depletion which can arise from such conditions as gastroenteritis, diabetic ketoacidosis, ileus, and ascites.

Chronic hyponatraemia arising from inappropriate secretion of antidiuretic hormone should ideally be corrected by fluid restriction. If sodium chloride is required for acute or chronic hyponatraemia, regardless of the cause, the deficit should be corrected slowly to avoid the risk of osmotic demyelination syndrome and the rise in plasma sodium concentration should not exceed 10 mmol/L in 24 hours. In severe hyponatraemia, sodium chloride 1.8 % may be used cautiously.

Hartmann's solution

Hartmann's solution (compound sodium lactate) can be used instead of isotonic sodium chloride solution during or after surgery, or in the initial management of the injured or wounded; it may reduce the risk of hyperchloraemic acidosis.

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
- HCO_3^- 29 mmol/L
- Cl^- 111 mmol/L
- Ca^{2+} 2 mmol/L

Glucose

Glucose solutions (5%) are used mainly to replace water deficit. Average water requirements in a healthy adult are 1.5 to 2.5 litres daily and this is needed to balance unavoidable losses of water through the skin and lungs and to provide sufficient for urinary excretion. Dehydration may occur when these losses are not compensated for by intake e.g. in coma, or in the elderly. Excessive loss of water without loss of electrolyte is uncommon, occurring in fevers, hyperthyroidism, diabetes insipidus and hypercalcaemia.

Glucose solutions are also used to correct and prevent hypoglycaemia and to provide a source of energy in those too ill to be fed adequately by mouth.

Glucose solutions are also given in regimens with calcium and insulin for the emergency management of hyperkalaemia. They are also given after correction of hyperglycaemia during treatment of diabetic ketoacidosis, when they must be accompanied by continuing insulin infusion.

Potassium chloride mixtures

Potassium chloride with sodium chloride intravenous infusion is the initial treatment for the correction of severe hypokalaemia and when sufficient potassium cannot be taken by mouth. Repeated measurement of plasma-potassium concentration is necessary to avoid the development of hyperkalaemia, particularly in renal impairment. Initial potassium replacement therapy should not involve glucose infusions, because glucose may cause a further decrease in the plasma-potassium concentration.

Sodium bicarbonate

Sodium bicarbonate is used to control severe metabolic acidosis (pH < 7.1) particularly that caused by loss of bicarbonate (as in renal tubular acidosis or from excessive gastrointestinal losses). Mild metabolic acidosis associated with volume depletion should first be managed by appropriate fluid replacement because acidosis usually resolves as tissue and renal perfusion are restored.

In more severe metabolic acidosis or when the acidosis remains unresponsive to correction of anoxia or hypovolaemia, sodium bicarbonate (1.26%) can be infused over 3 – 4 hours with plasma-pH and electrolyte monitoring. In severe shock, for example in cardiac arrest, metabolic acidosis can develop without sodium or volume depletion; in these circumstances sodium bicarbonate is best given as a small volume of hypertonic solution, such as 50 mL of 8.4% solution intravenously.

Albumin

Albumin solutions, prepared from whole blood, contain soluble proteins and electrolytes but no clotting factors, blood group antibodies, or plasma cholinesterases thus may be given without regard to the recipient's blood group.

Albumin is usually used after the acute phase of illness to correct a plasma-volume deficit; hypoalbuminaemia itself is not an appropriate indication and the use of albumin solution in acute plasma or blood loss is wasteful where plasma substitutes should be used instead.

Concentrated albumin solution (20%) can be used under specialist supervision in patients with an intravascular fluid deficit and oedema because of interstitial fluid overload, to restore intravascular plasma volume with less exacerbation of the salt and water overload than isotonic solutions. Concentrated albumin solution may also be used to obtain a diuresis in hypoalbuminaemic patients (e.g. in hepatic cirrhosis).

Plasma substitutes

Dextran and gelatin may be used at the outset to expand and maintain blood volume in shock arising from conditions such as burns or septicaemia; they may also be used as an immediate short-term measure to treat haemorrhage until blood is available. Dextran and gelatin are rarely needed when shock is due to sodium and water depletion because, in these circumstances, the shock responds to water and electrolyte repletion.

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Pharmacology: Gastro, Fluids and MSK

Question 28 of 49

Regarding antacids, which of the following statements is CORRECT:

a Antacids act to inhibit H2-receptors on gastric parietal cells.

b Antacids should not be taken at the same time as other drugs as they impair absorption.

c Antacids are first line for treatment of peptic ulcer disease.

d Aluminium hydroxide tends to cause diarrhoea.

e Antacids are contraindicated in hypocalcaemia.

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Pharmacology: Gastro, Fluids and MSK



Question 28 of 49

Regarding antacids, which of the following statements is CORRECT:

a) **Antacids act to inhibit H2-receptors on gastric parietal cells.** ✗
b) Antacids should not be taken at the same time as other drugs as they impair absorption. ✓
c) Antacids are first line for treatment of peptic ulcer disease.
d) Aluminium hydroxide tends to cause diarrhoea.
e) Antacids are contraindicated in hypocalcaemia.

Answer

Antacids should preferably not be taken at the same time as other drugs since they may impair absorption. Antacids act by neutralising stomach acid. They are used for symptomatic relief in dyspepsia, but are not first line for proven peptic ulcer disease where anti-secretory drugs have a better healing effect. Magnesium-containing antacids tend to be laxative whereas aluminium-containing antacids tend to be constipating. Antacids are contraindicated in hypophosphataemia.

Notes

Dyspepsia is upper abdominal discomfort or pain which may be described as a burning sensation, a heaviness, or an ache. It is often related to eating and may be accompanied by other symptoms such as nausea, fullness in the upper abdomen, or belching. The most common causes of dyspepsia are gastro-oesophageal reflux disease (GORD), peptic ulcer disease and non-ulcer (functional) dyspepsia.

Patients with dyspepsia/GORD should be advised that symptoms may improve if they:

- lose weight (if they are overweight)
- stop smoking
- reduce alcohol consumption
- reduce intake of any food or drink associated with worsening symptoms
- avoid having meals within 3 – 4 hours of going to bed
- raise the height of the head of the bed by a few inches

Antacids

Antacids are used for symptomatic relief in dyspepsia and in GORD.

Mechanism of action

Antacids work by neutralising gastric acid and raising the luminal pH. However, the effect is only for a relatively short period, requiring frequent administration. Antacids do promote ulcer healing, but less well than anti-secretory drugs.

Administration

Antacids are best taken when symptoms occur or are expected, usually between meals and at bedtime. Liquid preparations are more effective than tablet preparations.

Interactions

Antacids should preferably not be taken at the same time as other drugs since they may impair absorption. Antacids may damage enteric coatings designed to prevent dissolution in the stomach.

Side effects

Aluminium- and magnesium-containing antacids (e.g. aluminium hydroxide, magnesium carbonate), being relatively insoluble in water, are long acting if retained in the stomach. Magnesium-containing antacids tend to be laxative whereas aluminium-containing antacids tend to be constipating; antacids containing both magnesium and aluminium may reduce these colonic side effects.

Contraindications

Antacids are contraindicated in hypophosphataemia.

Alginates

Alginates taken in combination with an antacid increases the viscosity of stomach contents and can protect the oesophageal mucosa from acid reflux. Some alginate-containing preparations e.g. Gaviscon form a viscous gel raft that floats on the surface of the stomach contents, thereby reducing symptoms of reflux.

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Pharmacology: Gastro, Fluids and MSK

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You wish to prescribe an antimuscarinic drug to your patient. Which of the following is a contraindication:

a Parkinson disease

b Hypophosphatemia

c Prostatic enlargement

d Asthma

e Hypothyroidism

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Question 29 of 49

You wish to prescribe an antimuscarinic drug to your patient. Which of the following is a contraindication:

a) **Parkinson disease** ✖
b) Hypophosphatemia
c) Prostatic enlargement ✔
d) Asthma
e) Hypothyroidism

Answer

Antimuscarinics are contraindicated in:

- gastrointestinal obstruction
- intestinal atony
- myasthenia gravis
- paralytic ileus
- prostatic enlargement
- pyloric stenosis
- severe ulcerative colitis
- significant bladder outflow obstruction
- toxic megacolon
- urinary retention

Notes

Antimuscarinics

Antimuscarinics reduce intestinal motility (by acting on acetylcholine receptors and relaxing smooth muscle) and can be used as antispasmodics for the management of irritable bowel syndrome. Hyoscine butylbromide is advocated as a gastrointestinal antispasmodic, but it is poorly absorbed and thus has limited clinical utility.

Other indications for antimuscarinic drugs include arrhythmias, asthma and airways disease, motion sickness, parkinsonism, urinary incontinence, mydriasis and cycloplegia, premedication, and as an antidote to organophosphate poisoning.

Cautions

Antimuscarinics should be used with caution in:

- acute myocardial infarction
- arrhythmias (may be worsened)
- autonomic neuropathy
- cardiac insufficiency (due to association with tachycardia)
- cardiac surgery (due to association with tachycardia)
- children (increased risk of side effects)
- conditions characterised by tachycardia
- congestive heart failure (may be worsened)
- coronary artery disease (may be worsened)
- diarrhoea
- elderly (especially if frail)
- gastro-oesophageal reflux disease
- hiatus hernia with reflux oesophagitis
- hypertension
- hyperthyroidism (due to association with tachycardia)
- individuals susceptible to angle-closure glaucoma
- prostatic hyperplasia
- pyrexia
- ulcerative colitis

Contraindications

Antimuscarinics are contraindicated in:

- gastrointestinal obstruction
- intestinal atony
- myasthenia gravis
- paralytic ileus
- prostatic enlargement
- pyloric stenosis
- severe ulcerative colitis
- significant bladder outflow obstruction
- toxic megacolon
- urinary retention

Side effects

Many drugs have antimuscarinic effects; concomitant use of two or more such drugs can increase side effects such as dry mouth, urine retention, and constipation.

Side effects of antimuscarinics include:

- dilation of pupils with loss of accommodation (cycloplegia) and photophobia resulting in blurred vision ("Blind as a bat")
- dry mouth, eyes and skin ("Dry as a bone")
- elevated temperature ("Hot as a hare")
- skin flushing ("Red as a beet")
- confusion or agitation particularly in the elderly ("Mad as a hatter")
- reduced bronchial secretions
- transient bradycardia followed by tachycardia, palpitation and arrhythmias
- urinary retention
- constipation

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Pharmacology: Gastro, Fluids and MSK

Question 30 of 49

A 64 year old man, with a history of chronic alcohol abuse, presents to ED in alcohol withdrawal as he ran out of beer money. He is unkempt and malnourished. The alcohol nurse is concerned about his risk of Wernicke-Korsakoff syndrome. Which of the following would be most appropriate to prescribe this patient:

a Oral thiamine

b Intramuscular hydroxocobalamin

c Intravenous Pabrinex

d Oral ascorbic acid

e Oral ferrous sulfate

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Pharmacology: Gastro, Fluids and MSK

Question 31 of 49

A 69 year old man presents to hospital with an acutely painful, red, hot swollen big toe. He has a past medical history of gout, diabetes mellitus and severe heart failure. Gout is diagnosed. Which of the following is the drug of choice in this patient:

a NSAID

b Allopurinol

c Prednisolone

d Colchicine

e Paracetamol

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Pharmacology: Gastro, Fluids and MSK



Question 31 of 49

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a) NSAID ✖
b) Allopurinol
c) Prednisolone
d) Colchicine ✔
e) Paracetamol

Answer

NSAIDs are contraindicated in this patient due to his severe heart failure. Colchicine is a useful alternative in patients in whom NSAIDs are contraindicated, not tolerated or ineffective. Although its use is limited by toxicity at higher doses, it is useful in patients with heart failure or those taking anticoagulants. The dose is 500 micrograms 2 – 4 times a day until the symptoms are relieved or diarrhoea or vomiting occurs (max 6 mg per course and do not repeat treatment within 3 days).

Notes

Gout is a disorder of purine metabolism characterised by a raised uric acid level in the blood (hyperuricaemia) and the deposition of urate crystals in joints and other tissues, such as soft connective tissues or the urinary tract.

NSAIDs

Acute attacks of gout are usually treated with an NSAID such as diclofenac, indometacin, or naproxen. Treatment should be started as soon as possible and continued until 48 hours after the attack has resolved. A PPI should be co-prescribed for gastric protection in people at high risk of gastrointestinal adverse events.

Colchicine

Colchicine is an alternative in patients in whom NSAIDs are contraindicated, not tolerated or ineffective. Although its use is limited by toxicity at higher doses, it is useful in patients with heart failure or those taking anticoagulants.

The dose is 500 micrograms 2 – 4 times a day until the symptoms are relieved or diarrhoea or vomiting occurs (max 6 mg per course and do not repeat treatment within 3 days).

Colchicine should be avoided in people with blood dyscrasias and bone marrow disease.

Common side effects of colchicine include:

- abdominal pain
- nausea and vomiting
- diarrhoea

Side effects are dose dependent and may be severe enough to limit treatment.

Corticosteroids

Oral or parenteral corticosteroids are an effective alternative in those who cannot tolerate or who are resistant to NSAIDs and colchicine. Intra-articular injection of a corticosteroid can be used occasionally in acute monoarticular gout.

Allopurinol

Allopurinol is not effective in treating an acute attack and may prolong it indefinitely if started during the acute episode, but should be continued during an acute attack if the patient is already established on long term therapy.

Long term treatment (prevention) of gout may be indicated in patients with frequent recurrence of acute attacks of gout, the presence of tophi, or signs of chronic gouty arthritis. Allopurinol reduces uric acid formation from purines and may be used to prevent further attacks of gout by correcting hyperuricaemia. The initiation of treatment may precipitate an acute attack, and should be covered with an NSAID or colchicine, continued for at least one month after the hyperuricaemia has been corrected.

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Pharmacology: Gastro, Fluids and MSK

Question 32 of 49

Which of the following drugs used in the management of hyperkalaemia does not affect serum potassium levels:

a Insulin

b Calcium resonium

c Calcium gluconate

d Salbutamol

e Sodium bicarbonate

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Pharmacology: Gastro, Fluids and MSK



Question 32 of 49

Which of the following drugs used in the management of hyperkalaemia does not affect serum potassium levels:

a) Insulin
b) **Calcium resonium** ✗
c) Calcium gluconate ✓
d) Salbutamol
e) Sodium bicarbonate

Answer

Calcium gluconate is given to antagonise cardiac cell membrane excitability to reduce the risk of arrhythmias. It has no effect on serum potassium levels unlike the alternative drugs listed above.

Notes

Acute severe hyperkalaemia is characterised by a plasma-potassium concentration above 6.5 mmol/litre or the presence of ECG changes. ECG changes observed in hyperkalaemia may include tall peaked T waves, flattening or loss of P waves, broadening of QRS complexes, PR prolongation and bradycardia.

Emergency management

1. Reduce cardiac cell membrane excitability:
   - **10 ml calcium gluconate 10% by slow intravenous injection over 5 minutes**
     - ECG changes should improve within 1 – 3 minutes, but only has a transient effect of about 30 minutes
     - If there is no improvement, a further 10 ml should be given every 5 – 10 minutes until the ECG normalises (max 3 doses in total)
     - If the patient is taking digoxin, the calcium gluconate should be given slowly (mixed with 100 ml 5% glucose and given over 20 minutes) as rapid calcium administration may precipitate myocardial digoxin toxicity
2. Transcellular shift from extracellular to intracellular space:
   - **Intravenous infusion of insulin-glucose given as 10 units of Actrapid added to 50 ml of glucose 50% infused over 30 minutes**
     - Potassium should decrease by about 0.6 – 1.0 mmol/L in 15 minutes and the reduction lasts for about 60 minutes
     - This is repeated if necessary or a continuous infusion initiated
   - **Nebulised salbutamol 10 mg**
     - Reduces potassium by about 0.5 – 1.0 mmol/L in 15 – 30 minutes and lasts for about two hours
     - Should be used with caution in patients with ischaemic heart disease and history of cardiac arrhythmias
     - Response is attenuated in patients on beta-blockers and digoxin
   - The correction of causal or compounding acidosis with sodium bicarbonate infusion should be considered (if pH < 7.2)
     - Correction of acidosis promotes intracellular uptake of K+
3. Reduce total body potassium:
   - Any potassium supplements should be stopped, as well as any drugs that might precipitate hyperkalaemia (e.g. digoxin, beta-blockers)
   - Consider low potassium diet
   - Urinary potassium loss should be promoted with the use of appropriate fluids or diuretics to maintain good urine output
   - **Calcium resonium 15 g PO TDS**
     - Ion-exchange resin that exchanges sodium for potassium as it passes through the intestine
     - Should be given with regular laxatives to prevent constipation
     - Each gram removes approximately 1 mmol/L of potassium but onset is slow, taking 2 – 6 hours or longer
     - Contraindicated in patients with pre-existing hypercalcaemia

If the patient does not respond to the above measures dialysis will be required. DIalysis is likely to be needed if potassium is very high (> 7.5 mmol/L), if the patient is oligo/anuric, if the patient is already on long term dialysis or if the patient has advanced CKD.

Treatment	Administration	Mechanism of Action	Time to Onset of Action	Duration of Action	Achievable reduction of serum K+
Calcium resonium	15 g PO TDS	Ion-exchange resin that exchanges sodium for potassium as it passes through intestine	2 – 6 hours or longer	4 – 6 hours	Unknown
Calcium gluconate	10 ml calcium gluconate 10% by IV injection over 5 mins	Antagonises cardiac membrane excitability	1 – 3 minutes	30 minutes	N/A
Insulin-glucose infusion	10 units Actrapid in 50 ml glucose 50% infused over 30 mins	Increases intracellular uptake of K+ via Na-K ATP pump	Within 15 minutes	60 minutes	0.6 – 1 mmol/L
Nebulised salbutamol	10 mg nebulised	Increases intracellular uptake of K+ via Na-K ATP pump	Within 30 minutes	1 – 3 hours	0.5 – 1 mmol/L
Sodium bicarbonate	1.4% 500 ml IV over 2 hours	Corrects acidosis and thus promotes intracellular uptake of K+	After 60 minutes	Unknown	Unknown

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Pharmacology: Gastro, Fluids and MSK

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Antispasmodic drugs are primarily indicated in which of the following conditions:

a Bowel obstruction

b Inflammatory bowel disease

c Urinary retention

d Gastroenteritis

e Irritable bowel syndrome

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Antispasmodic drugs are primarily indicated in which of the following conditions:

a) **Bowel obstruction** ✗
b) Inflammatory bowel disease
c) Urinary retention
d) Gastroenteritis
e) Irritable bowel syndrome ✓

Answer

Antispasmodics, such as antimuscarinics, may be used in the management of irritable bowel syndrome. Other antispasmodics used include direct-acting smooth muscle relaxants such as mebeverine, alverine, and peppermint oil. Antispasmodics are contraindicated in bowel obstruction and severe inflammatory bowel disease. Antispasmodics are occasionally of value in treating abdominal cramp associated with diarrhoea but they should not be used for primary treatment. Antispasmodics should be avoided in young children with gastroenteritis because they are rarely effective and have troublesome side effects. Antimuscarinics are contraindicated in urinary retention.

Notes

Antimuscarinics

Antimuscarinics reduce intestinal motility (by acting on acetylcholine receptors and relaxing smooth muscle) and can be used as antispasmodics for the management of irritable bowel syndrome. Hyoscine butylbromide is advocated as a gastrointestinal antispasmodic, but it is poorly absorbed and thus has limited clinical utility.

Other indications for antimuscarinic drugs include arrhythmias, asthma and airways disease, motion sickness, parkinsonism, urinary incontinence, mydriasis and cycloplegia, premedication, and as an antidote to organophosphate poisoning.

Cautions

Antimuscarinics should be used with caution in:

- acute myocardial infarction
- arrhythmias (may be worsened)
- autonomic neuropathy
- cardiac insufficiency (due to association with tachycardia)
- cardiac surgery (due to association with tachycardia)
- children (increased risk of side effects)
- conditions characterised by tachycardia
- congestive heart failure (may be worsened)
- coronary artery disease (may be worsened)
- diarrhoea
- elderly (especially if frail)
- gastro-oesophageal reflux disease
- hiatus hernia with reflux oesophagitis
- hypertension
- hyperthyroidism (due to association with tachycardia)
- individuals susceptible to angle-closure glaucoma
- prostatic hyperplasia
- pyrexia
- ulcerative colitis

Contraindications

Antimuscarinics are contraindicated in:

- gastrointestinal obstruction
- intestinal atony
- myasthenia gravis
- paralytic ileus
- prostatic enlargement
- pyloric stenosis
- severe ulcerative colitis
- significant bladder outflow obstruction
- toxic megacolon
- urinary retention

Side effects

Many drugs have antimuscarinic effects; concomitant use of two or more such drugs can increase side effects such as dry mouth, urine retention, and constipation.

Side effects of antimuscarinics include:

- dilation of pupils with loss of accommodation (cycloplegia) and photophobia resulting in blurred vision ("Blind as a bat")
- dry mouth, eyes and skin ("Dry as a bone")
- elevated temperature ("Hot as a hare")
- skin flushing ("Red as a beet")
- confusion or agitation particularly in the elderly ("Mad as a hatter")
- reduced bronchial secretions
- transient bradycardia followed by tachycardia, palpitation and arrhythmias
- urinary retention
- constipation

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Pharmacology: Gastro, Fluids and MSK

Question 34 of 49

Regarding proton pump inhibitors, which of the following statements is CORRECT:

a They are used alone for Helicobacter pylori eradication therapy.

b They are protective against gastrointestinal infections.

c They should not be used concomitantly with NSAID therapy.

d Intravenous PPI following endoscopic treatment reduces the risk of rebleeding in a severe peptic ulcer bleed.

e Prolonged therapy with PPIs may cause hypermagnesaemia.

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Pharmacology: Gastro, Fluids and MSK

Question 34 of 49

Regarding proton pump inhibitors, which of the following statements is CORRECT:

a) **They are used alone for Helicobacter pylori eradication therapy.** ✖

b) They are protective against gastrointestinal infections.

c) They should not be used concomitantly with NSAID therapy.

d) ✔

Intravenous PPI following endoscopic treatment reduces the risk of rebleeding in a severe peptic ulcer bleed.

e) Prolonged therapy with PPIs may cause hypermagnesaemia.

Answer

Proton pump inhibitors act by directly inhibiting the H^+/K^+ ATPase (proton) pump on gastric parietal cell luminal membranes and thus inhibiting acid secretion.

Notes

Mechanism of action

An intravenous high dose PPI reduces the risk of rebleeding and the need for surgery when used for post-endoscopic treatment of severe peptic ulcer bleeding. Prolonged PPI therapy is associated with hypomagnesaemia. PPIs are associated with an increased risk of gastrointestinal infection (including C. diff infection). PPIs are used in combination with antibiotics for H. pylori eradication therapy. PPIs are used for treatment and prevention of NSAID-associated ulceration.

Indications

Proton pump inhibitors may be indicated for:

- treatment of gastric and duodenal ulcers
- eradication of Helicobacter pylori in combination with antibacterials
- treatment of dyspepsia and GORD
- post-endoscopic treatment of severe peptic ulcer bleeding (an intravenous high dose PPI reduces risk of rebleeding and the need for surgery)
- treatment and prevention of NSAID-associated ulcers
- cystic fibrosis (reduces degradation of pancreatic enzyme supplements)
- Zollinger-Ellison syndrome (controls excessive gastric acid secretion)

Cautions

Proton pump inhibitors:

- can increase the risk of fractures (particularly when used at high doses for over a year in the elderly or used in those at risk of osteoporosis)
- may increase the risk of gastrointestinal infections (including Clostridium difficile infection)
- may mask the symptoms of gastric cancer (in patients with 'alarm features', gastric malignancy should be ruled out before starting treatment)
- can cause false negative H. pylori test results (and should be stopped at least 2 weeks before testing)

Side effects

Common side effects (usually mild and reversible) include:

- abdominal pain
- constipation
- diarrhoea
- flatulence
- headache
- nausea
- vomiting
- dizziness
- skin rashes

Possible adverse effects of long-term PPI treatment:

- Hypomagnesaemia (measurement of serum magnesium concentrations should be considered before and during prolonged treatment with a PPI, especially when used with other drugs that cause hypomagnesaemia or with digoxin)
- Rebound acid hypersecretion and protracted dyspepsia after stopping prolonged treatment
- Interstitial nephritis
- Vitamin B12 deficiency

Administration

Omeprazole:

- H. pylori eradication regimen dose: 20 mg od
- Healing peptic ulcer: 20 mg od (40 mg od may be used in severe or recurrent cases)
- Healing NSAID-associated ulcer: 20 mg od
- Prophylaxis for NSAID-associated ulcer: 20 mg od
- Major peptic ulcer bleeding (following endoscopic treatment): Initially 80 mg, to be given over 40–60 minutes, then (by continuous intravenous infusion) 8 mg/hour for 72 hours, subsequent dose then changed to oral therapy

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Proton pump inhibitors act by inhibiting which of the following:

a H^+ ATPase pump

b Na^+/K^+ ATPase pump

c H^+/Cl^- symporter

d H^+/K^+ ATPase pump

e Na^+/H^+ ATPase pump

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Proton pump inhibitors act by inhibiting which of the following:

a) H^+ ATPase pump ✖
b) Na^+/K^+ ATPase pump
c) H^+/Cl^- symporter
d) H^+/K^+ ATPase pump ✔
e) Na^+/H^+ ATPase pump

Answer

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Notes

Mechanism of action

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Indications

Proton pump inhibitors may be indicated for:

- treatment of gastric and duodenal ulcers
- eradication of Helicobacter pylori in combination with antibacterials
- treatment of dyspepsia and GORD
- post-endoscopic treatment of severe peptic ulcer bleeding (an intravenous high dose PPI reduces risk of rebleeding and the need for surgery)
- treatment and prevention of NSAID-associated ulcers
- cystic fibrosis (reduces degradation of pancreatic enzyme supplements)
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Pharmacology: Gastro, Fluids and MSK

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Regarding diclofenac, which of the following is CORRECT:

a It has a greater efficacy than both ibuprofen and naproxen.

b It has more side effects than ibuprofen but fewer than naproxen.

c It has a lower efficacy but less side effects than naproxen.

d It has a lower efficacy than naproxen with more side effects.

e It has a similar efficacy to naproxen.

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Pharmacology: Gastro, Fluids and MSK

Question 36 of 49

Regarding diclofenac, which of the following is CORRECT:

a) It has a greater efficacy than both ibuprofen and naproxen. ✖
b) It has more side effects than ibuprofen but fewer than naproxen.
c) It has a lower efficacy but less side effects than naproxen.
d) It has a lower efficacy than naproxen with more side effects.
e) It has a similar efficacy to naproxen. ✔

Answer

Naproxen has good efficacy with a relatively low incidence of side effects (but more than ibuprofen); it is recommended first line treatment in acute gout. Diclofenac is similar in efficacy to naproxen but is associated with a higher incidence of cardiovascular adverse events (and is in fact contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure).

Notes

Mechanism of action

NSAIDs reduce the production of prostaglandins by inhibiting the cyclo-oxygenase (COX) enzymes with analgesic, antipyretic and anti-inflammatory effects. The two main types of COX enzyme are COX-1 (which produces prostaglandins that help to maintain gastric mucosal integrity and platelet-initiated blood clotting) and COX-2 (which produces prostaglandins that mediate pain and inflammation). NSAIDs vary in their selectivity for inhibiting different types of COX; specific inhibition of COX-2 (e.g. celecoxib and etoricoxib) is associated with less gastrointestinal intolerance.

Therapeutic effects

In single doses NSAIDs have analgesic activity comparable to that of paracetamol, therefore given their side effect profile, paracetamol is preferred, particularly in the elderly. In regular full dosage NSAIDs have both a lasting analgesic and an anti-inflammatory effect making them useful for the management of continuous or regular pain associated with inflammation e.g. inflammatory arthritides. Pain relief starts soon after taking the first dose and a full analgesic effect should normally be obtained within a week, whereas an anti-inflammatory effect may not be achieved (or may not be clinically assessable) for up to 3 weeks.

Choice of NSAID

Ibuprofen has fewer side effects than other non-selective NSAIDs but its anti-inflammatory properties are weaker, making it unsuitable for conditions where inflammation is prominent e.g. acute gout.

Naproxen has good efficacy with a relatively low incidence of side effects (but more than ibuprofen); it is recommended first line treatment in acute gout.

Diclofenac is similar in efficacy to naproxen but is associated with a higher incidence of cardiovascular adverse events (and is in fact contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure).

Mefenamic acid has only minor anti-inflammatory properties and is typically used in the management of painful dysmenorrhoea. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment.

Risk of NSAID-induced GI adverse events	NSAIDs
Lowest risk	Ibuprofen
Intermediate risk	Naproxen, diclofenac, indometacin
Highest risk	Piroxicam, ketoprofen, ketorolac

Contraindications

NSAIDs are contraindicated in people with:

- A history of hypersensitivity or severe allergic reaction to aspirin or any other NSAID
- Severe heart failure (NSAIDs may impair renal function)
- Liver fibrosis, cirrhosis or acute liver failure (risk of variceal bleeding, hepatorenal syndrome and encephalopathy)
- Severe hepatic impairment (e.g. liver enzyme levels more than three times the upper limit of the normal range; serum albumin less than 25 g/L)
- Current treatment for gastrointestinal bleeding, symptomatic peptic ulcer, or gastrointestinal perforation or obstruction
- Coxibs and diclofenac are also contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, mild, moderate, or severe heart failure
- Renal failure, with estimated glomerular filtration rate (eGFR) less than 30–15 mL/min/1.73 m2, or creatinine clearance less than 30–20 mL/min, or dehydration (relative)

Cautions

NSAIDs should be used with caution in:

- People with asthma (patients should be offered a trial to assess effect on asthma control)
- The elderly (increased risk of serious adverse effects such as gastrointestinal bleeding and perforation, which may be fatal)
- People with a history of peptic ulceration (standard NSAIDs contraindicated), or those at high risk of gastrointestinal adverse effects
- People with inflammatory bowel disease (NSAIDs may increase the risk of developing or cause exacerbations of ulcerative colitis or Crohn's disease)
- People with hepatitis or cholestasis (increased risk of gastrointestinal bleeding and fluid retention)
- Renal impairment (avoid if possible, sodium and water retention may occur leading to a deterioration in renal function and, possibly renal failure)
- People with heart failure (NSAIDs may impair renal function)
- People with hypertension (NSAIDs may impair renal function)
- In a women trying to conceive (NSAIDs may impair female fertility)

Adverse effects

Adverse effects may be minimised by selecting an appropriate NSAID and using the lowest effective dose for the shortest duration necessary to control symptoms.

Side effects of NSAIDs include:

- The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications such as ulcer, perforation, obstruction or bleeding (especially if risk factors are present – see below)
- Less commonly NSAIDs may cause cardiovascular and renal complications such as myocardial infarction, stroke, cardiac failure, hypertension, and renal failure (the risk is increased in people with IHD, CVD, PAD, CKD, HF or HTN, risk factors for cardiovascular disease and people > 65 years)
- Prolonged bleeding (for example after surgery) because of platelet inhibition
- Asthma (NSAIDs may exacerbate or precipitate asthma. Stop NSAID if it is suspected to have precipitated bronchospasm)
- Severe skin reactions and angioedema (stop NSAID)
- Very rarely, NSAIDs can precipitate severe hepatic reactions such as hepatitis, liver necrosis, or hepatic failure (stop NSAID)

Risk factors for increased risk of gastrointestinal adverse effects

People are at high risk of serious NSAID–induced gastrointestinal adverse events if they have one or more of the following risk factors:

- Using the maximum recommended dose of an NSAID
- Aged 65 years or older
- History of gastroduodenal ulcer, GI bleeding, or gastroduodenal perforation
- Concomitant use of medications that are known to increase the likelihood of upper GI adverse events (e.g. anticoagulants, aspirin, corticosteroids, and antidepressants (selective serotonin reuptake inhibitors, venlafaxine, or duloxetine))
- Serious comorbidity, such as cardiovascular disease, hepatic or renal impairment, diabetes, or hypertension
- Requirement for prolonged NSAID use including people with osteoarthritis or rheumatoid arthritis of any age and people aged 45 years or older with chronic low back pain
- The presence of Helicobacter pylori infection
- Excessive alcohol use
- Heavy smoking

Prevention and management of gastrointestinal adverse effects

- Preventing gastrointestinal adverse effects
  - Consider prescribing:
    - An alternative to an NSAID e.g. paracetamol +/- codeine
    - The lowest dose of NSAID for the shortest period of time
    - Only one NSAID at a time (avoid concomitant use with aspirin)
    - A COX-2 selective NSAID (but increased cardiovascular risk)
  - If the person is at an increased risk of GI adverse events and requires an NSAID, co-prescribe a proton pump inhibitor (PPI) for gastroprotection (e.g. omeprazole 20 mg od, lansoprazole 15 – 30 mg od)
- Managing gastrointestinal adverse effects
  - Where possible the NSAID should be withdrawn if an ulcer occurs
  - The ulcer should be treated with a PPI (a H2-receptor antagonist or misoprostol are alternatives)
  - On healing the patient should be tested for H. pylori and treated if required
  - If treatment with NSAID needs to continue, options include:
    - On healing continue PPI
    - Switch to selective COX-2 inhibitor

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Pharmacology: Gastro, Fluids and MSK

Question 37 of 49

Intravenous sodium bicarbonate is used primarily for which of the following:

a Severe metabolic acidosis

b Severe peptic ulcer bleeding

c Beta blocker overdose

d Severe hyperkalaemia

e Severe hyponatraemia

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Pharmacology: Gastro, Fluids and MSK



Question 37 of 49

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a) Severe metabolic acidosis ✔
b) Severe peptic ulcer bleeding
c) Beta blocker overdose
d) Severe hyperkalaemia
e) Severe hyponatraemia

Answer

Sodium bicarbonate is used to control severe metabolic acidosis (pH < 7.1) particularly that caused by loss of bicarbonate (as in renal tubular acidosis or from excessive gastrointestinal losses).

Notes

Solutions of electrolytes are given intravenously to meet normal fluid and electrolyte requirements or to replenish substantial deficits or continuing losses when the patient is nauseated or vomiting and is unable to take adequate amounts by mouth. When intravenous administration is not possible, fluid (as sodium chloride 0.9% or glucose 5%) can also be given by subcutaneous infusion.

Sodium chloride

Normal saline (sodium chloride 0.9%) contains:

- Na^+ 150 mmol/L
- Cl^- 150 mmol/L

Sodium chloride in isotonic solution (0.9%) provides the most important extracellular ions in near physiological concentrations and is indicated in sodium depletion which can arise from such conditions as gastroenteritis, diabetic ketoacidosis, ileus, and ascites.

Chronic hyponatraemia arising from inappropriate secretion of antidiuretic hormone should ideally be corrected by fluid restriction. If sodium chloride is required for acute or chronic hyponatraemia, regardless of the cause, the deficit should be corrected slowly to avoid the risk of osmotic demyelination syndrome and the rise in plasma sodium concentration should not exceed 10 mmol/L in 24 hours. In severe hyponatraemia, sodium chloride 1.8 % may be used cautiously.

Hartmann's solution

Hartmann's solution (compound sodium lactate) can be used instead of isotonic sodium chloride solution during or after surgery, or in the initial management of the injured or wounded; it may reduce the risk of hyperchloraemic acidosis.

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
- HCO_3^- 29 mmol/L
- Cl^- 111 mmol/L
- Ca^{2+} 2 mmol/L

Glucose

Glucose solutions (5%) are used mainly to replace water deficit. Average water requirements in a healthy adult are 1.5 to 2.5 litres daily and this is needed to balance unavoidable losses of water through the skin and lungs and to provide sufficient for urinary excretion. Dehydration may occur when these losses are not compensated for by intake e.g. in coma, or in the elderly. Excessive loss of water without loss of electrolyte is uncommon, occurring in fevers, hyperthyroidism, diabetes insipidus and hypercalcaemia.

Glucose solutions are also used to correct and prevent hypoglycaemia and to provide a source of energy in those too ill to be fed adequately by mouth.

Glucose solutions are also given in regimens with calcium and insulin for the emergency management of hyperkalaemia. They are also given after correction of hyperglycaemia during treatment of diabetic ketoacidosis, when they must be accompanied by continuing insulin infusion.

Potassium chloride mixtures

Potassium chloride with sodium chloride intravenous infusion is the initial treatment for the correction of severe hypokalaemia and when sufficient potassium cannot be taken by mouth. Repeated measurement of plasma-potassium concentration is necessary to avoid the development of hyperkalaemia, particularly in renal impairment. Initial potassium replacement therapy should not involve glucose infusions, because glucose may cause a further decrease in the plasma-potassium concentration.

Sodium bicarbonate

Sodium bicarbonate is used to control severe metabolic acidosis (pH < 7.1) particularly that caused by loss of bicarbonate (as in renal tubular acidosis or from excessive gastrointestinal losses). Mild metabolic acidosis associated with volume depletion should first be managed by appropriate fluid replacement because acidosis usually resolves as tissue and renal perfusion are restored.

In more severe metabolic acidosis or when the acidosis remains unresponsive to correction of anoxia or hypovolaemia, sodium bicarbonate (1.26%) can be infused over 3 – 4 hours with plasma-pH and electrolyte monitoring. In severe shock, for example in cardiac arrest, metabolic acidosis can develop without sodium or volume depletion; in these circumstances sodium bicarbonate is best given as a small volume of hypertonic solution, such as 50 mL of 8.4% solution intravenously.

Albumin

Albumin solutions, prepared from whole blood, contain soluble proteins and electrolytes but no clotting factors, blood group antibodies, or plasma cholinesterases thus may be given without regard to the recipient's blood group.

Albumin is usually used after the acute phase of illness to correct a plasma-volume deficit; hypoalbuminaemia itself is not an appropriate indication and the use of albumin solution in acute plasma or blood loss is wasteful where plasma substitutes should be used instead.

Concentrated albumin solution (20%) can be used under specialist supervision in patients with an intravascular fluid deficit and oedema because of interstitial fluid overload, to restore intravascular plasma volume with less exacerbation of the salt and water overload than isotonic solutions. Concentrated albumin solution may also be used to obtain a diuresis in hypoalbuminaemic patients (e.g. in hepatic cirrhosis).

Plasma substitutes

Dextran and gelatin may be used at the outset to expand and maintain blood volume in shock arising from conditions such as burns or septicaemia; they may also be used as an immediate short-term measure to treat haemorrhage until blood is available. Dextran and gelatin are rarely needed when shock is due to sodium and water depletion because, in these circumstances, the shock responds to water and electrolyte repletion.

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Pharmacology: Gastro, Fluids and MSK

Question 38 of 49

Hartmann's solution contains how much potassium:

a 1 mmol/L

b 5 mmol/L

c 11 mmol/L

d 2 mmol/L

e 29 mmol/L

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Pharmacology: Gastro, Fluids and MSK



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Chronic hyponatraemia arising from inappropriate secretion of antidiuretic hormone should ideally be corrected by fluid restriction. If sodium chloride is required for acute or chronic hyponatraemia, regardless of the cause, the deficit should be corrected slowly to avoid the risk of osmotic demyelination syndrome and the rise in plasma sodium concentration should not exceed 10 mmol/L in 24 hours. In severe hyponatraemia, sodium chloride 1.8 % may be used cautiously.

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Plasma substitutes

Dextran and gelatin may be used at the outset to expand and maintain blood volume in shock arising from conditions such as burns or septicaemia; they may also be used as an immediate short-term measure to treat haemorrhage until blood is available. Dextran and gelatin are rarely needed when shock is due to sodium and water depletion because, in these circumstances, the shock responds to water and electrolyte repletion.

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Pharmacology: Gastro, Fluids and MSK

Question 39 of 49

Which of the following best describes the mechanism of action of nonsteroidal anti-inflammatory drugs (NSAIDs):

a Acetylcholinesterase inhibition

b Xanthine oxidase inhibition

c Lipoxygenase inhibition

d Cyclo-oxygenase (COX) inhibition

e Cyclic nucleotide phosphodiesterase inhibition

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Pharmacology: Gastro, Fluids and MSK



Question 39 of 49

Which of the following best describes the mechanism of action of nonsteroidal anti-inflammatory drugs (NSAIDs):

a) **Acetylcholinesterase inhibition** ✘
b) Xanthine oxidase inhibition
c) Lipoxygenase inhibition
d) Cyclo-oxygenase (COX) inhibition ✔
e) Cyclic nucleotide phosphodiesterase inhibition

Answer

NSAIDs reduce the production of prostaglandins by inhibiting the cyclo-oxygenase (COX) enzymes with analgesic, antipyretic and anti-inflammatory effects.

Notes

Mechanism of action

NSAIDs reduce the production of prostaglandins by inhibiting the cyclo-oxygenase (COX) enzymes with analgesic, antipyretic and anti-inflammatory effects. The two main types of COX enzyme are COX-1 (which produces prostaglandins that help to maintain gastric mucosal integrity and platelet-initiated blood clotting) and COX-2 (which produces prostaglandins that mediate pain and inflammation). NSAIDs vary in their selectivity for inhibiting different types of COX; specific inhibition of COX-2 (e.g. celecoxib and etoricoxib) is associated with less gastrointestinal intolerance.

Therapeutic effects

In single doses NSAIDs have analgesic activity comparable to that of paracetamol, therefore given their side effect profile, paracetamol is preferred, particularly in the elderly. In regular full dosage NSAIDs have both a lasting analgesic and an anti-inflammatory effect making them useful for the management of continuous or regular pain associated with inflammation e.g. inflammatory arthritides. Pain relief starts soon after taking the first dose and a full analgesic effect should normally be obtained within a week, whereas an anti-inflammatory effect may not be achieved (or may not be clinically assessable) for up to 3 weeks.

Choice of NSAID

Ibuprofen has fewer side effects than other non-selective NSAIDs but its anti-inflammatory properties are weaker, making it unsuitable for conditions where inflammation is prominent e.g. acute gout.

Naproxen has good efficacy with a relatively low incidence of side effects (but more than ibuprofen); it is recommended first line treatment in acute gout.

Diclofenac is similar in efficacy to naproxen but is associated with a higher incidence of cardiovascular adverse events (and is in fact contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure).

Mefenamic acid has only minor anti-inflammatory properties and is typically used in the management of painful dysmenorrhoea. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment.

Risk of NSAID-induced GI adverse events	NSAIDs
Lowest risk	Ibuprofen
Intermediate risk	Naproxen, diclofenac, indometacin
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Contraindications

NSAIDs are contraindicated in people with:

- A history of hypersensitivity or severe allergic reaction to aspirin or any other NSAID
- Severe heart failure (NSAIDs may impair renal function)
- Liver fibrosis, cirrhosis or acute liver failure (risk of variceal bleeding, hepatorenal syndrome and encephalopathy)
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NSAIDs should be used with caution in:

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- Renal impairment (avoid if possible, sodium and water retention may occur leading to a deterioration in renal function and, possibly renal failure)
- People with heart failure (NSAIDs may impair renal function)
- People with hypertension (NSAIDs may impair renal function)
- In a women trying to conceive (NSAIDs may impair female fertility)

Adverse effects

Adverse effects may be minimised by selecting an appropriate NSAID and using the lowest effective dose for the shortest duration necessary to control symptoms.

Side effects of NSAIDs include:

- The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications such as ulcer, perforation, obstruction or bleeding (especially if risk factors are present – see below)
- Less commonly NSAIDs may cause cardiovascular and renal complications such as myocardial infarction, stroke, cardiac failure, hypertension, and renal failure (the risk is increased in people with IHD, CVD, PAD, CKD, HF or HTN, risk factors for cardiovascular disease and people > 65 years)
- Prolonged bleeding (for example after surgery) because of platelet inhibition
- Asthma (NSAIDs may exacerbate or precipitate asthma. Stop NSAID if it is suspected to have precipitated bronchospasm)
- Severe skin reactions and angioedema (stop NSAID)
- Very rarely, NSAIDs can precipitate severe hepatic reactions such as hepatitis, liver necrosis, or hepatic failure (stop NSAID)

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People are at high risk of serious NSAID–induced gastrointestinal adverse events if they have one or more of the following risk factors:

- Using the maximum recommended dose of an NSAID
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- The presence of Helicobacter pylori infection
- Excessive alcohol use
- Heavy smoking

Prevention and management of gastrointestinal adverse effects

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  - Consider prescribing:
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    - The lowest dose of NSAID for the shortest period of time
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Pharmacology: Gastro, Fluids and MSK

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Regarding naproxen, which of the following is CORRECT:

a It has a greater efficacy than diclofenac.

b It has a lower efficacy than diclofenac.

c It has more side effects than diclofenac.

d It has a similar efficacy to diclofenac but more side effects.

e It has a similar efficacy to diclofenac and less side effects.

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Diclofenac is similar in efficacy to naproxen but is associated with a higher incidence of cardiovascular adverse events (and is in fact contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease and mild, moderate, or severe heart failure).

Mefenamic acid has only minor anti-inflammatory properties and is typically used in the management of painful dysmenorrhoea. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment.

Risk of NSAID-induced GI adverse events	NSAIDs
Lowest risk	Ibuprofen
Intermediate risk	Naproxen, diclofenac, indometacin
Highest risk	Piroxicam, ketoprofen, ketorolac

Contraindications

NSAIDs are contraindicated in people with:

- A history of hypersensitivity or severe allergic reaction to aspirin or any other NSAID
- Severe heart failure (NSAIDs may impair renal function)
- Liver fibrosis, cirrhosis or acute liver failure (risk of variceal bleeding, hepatorenal syndrome and encephalopathy)
- Severe hepatic impairment (e.g. liver enzyme levels more than three times the upper limit of the normal range; serum albumin less than 25 g/L)
- Current treatment for gastrointestinal bleeding, symptomatic peptic ulcer, or gastrointestinal perforation or obstruction
- Coxibs and diclofenac are also contraindicated in people with ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, mild, moderate, or severe heart failure
- Renal failure, with estimated glomerular filtration rate (eGFR) less than 30–15 mL/min/1.73 m2, or creatinine clearance less than 30–20 mL/min, or dehydration (relative)

Cautions

NSAIDs should be used with caution in:

- People with asthma (patients should be offered a trial to assess effect on asthma control)
- The elderly (increased risk of serious adverse effects such as gastrointestinal bleeding and perforation, which may be fatal)
- People with a history of peptic ulceration (standard NSAIDs contraindicated), or those at high risk of gastrointestinal adverse effects
- People with inflammatory bowel disease (NSAIDs may increase the risk of developing or cause exacerbations of ulcerative colitis or Crohn's disease)
- People with hepatitis or cholestasis (increased risk of gastrointestinal bleeding and fluid retention)
- Renal impairment (avoid if possible, sodium and water retention may occur leading to a deterioration in renal function and, possibly renal failure)
- People with heart failure (NSAIDs may impair renal function)
- People with hypertension (NSAIDs may impair renal function)
- In a women trying to conceive (NSAIDs may impair female fertility)

Adverse effects

Adverse effects may be minimised by selecting an appropriate NSAID and using the lowest effective dose for the shortest duration necessary to control symptoms.

Side effects of NSAIDs include:

- The most common adverse effects of NSAIDs are dyspepsia and other upper gastrointestinal complications such as ulcer, perforation, obstruction or bleeding (especially if risk factors are present – see below)
- Less commonly NSAIDs may cause cardiovascular and renal complications such as myocardial infarction, stroke, cardiac failure, hypertension, and renal failure (the risk is increased in people with IHD, CVD, PAD, CKD, HF or HTN, risk factors for cardiovascular disease and people > 65 years)
- Prolonged bleeding (for example after surgery) because of platelet inhibition
- Asthma (NSAIDs may exacerbate or precipitate asthma. Stop NSAID if it is suspected to have precipitated bronchospasm)
- Severe skin reactions and angioedema (stop NSAID)
- Very rarely, NSAIDs can precipitate severe hepatic reactions such as hepatitis, liver necrosis, or hepatic failure (stop NSAID)

Risk factors for increased risk of gastrointestinal adverse effects

People are at high risk of serious NSAID–induced gastrointestinal adverse events if they have one or more of the following risk factors:

- Using the maximum recommended dose of an NSAID
- Aged 65 years or older
- History of gastroduodenal ulcer, GI bleeding, or gastroduodenal perforation
- Concomitant use of medications that are known to increase the likelihood of upper GI adverse events (e.g. anticoagulants, aspirin, corticosteroids, and antidepressants (selective serotonin reuptake inhibitors, venlafaxine, or duloxetine))
- Serious comorbidity, such as cardiovascular disease, hepatic or renal impairment, diabetes, or hypertension
- Requirement for prolonged NSAID use including people with osteoarthritis or rheumatoid arthritis of any age and people aged 45 years or older with chronic low back pain
- The presence of Helicobacter pylori infection
- Excessive alcohol use
- Heavy smoking

Prevention and management of gastrointestinal adverse effects

- Preventing gastrointestinal adverse effects
  - Consider prescribing:
    - An alternative to an NSAID e.g. paracetamol +/- codeine
    - The lowest dose of NSAID for the shortest period of time
    - Only one NSAID at a time (avoid concomitant use with aspirin)
    - A COX-2 selective NSAID (but increased cardiovascular risk)
  - If the person is at an increased risk of GI adverse events and requires an NSAID, co-prescribe a proton pump inhibitor (PPI) for gastroprotection (e.g. omeprazole 20 mg od, lansoprazole 15 – 30 mg od)
- Managing gastrointestinal adverse effects
  - Where possible the NSAID should be withdrawn if an ulcer occurs
  - The ulcer should be treated with a PPI (a H2-receptor antagonist or misoprostol are alternatives)
  - On healing the patient should be tested for H. pylori and treated if required
  - If treatment with NSAID needs to continue, options include:
    - On healing continue PPI
    - Switch to selective COX-2 inhibitor

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Pharmacology: Gastro, Fluids and MSK

Question 41 of 49

Loperamide acts on which of the following types of receptors:

a Muscarinic receptors

b Opioid receptors

c Dopamine receptors

d Alpha receptors

e Beta receptors

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Pharmacology: Gastro, Fluids and MSK

Question 41 of 49

Loperamide acts on which of the following types of receptors:

a) Muscarinic receptors ✗
b) Opioid receptors ✓
c) Dopamine receptors
d) Alpha receptors
e) Beta receptors

Answer

Loperamide acts on opioid mu-receptors inhibiting acetylcholine release from the myenteric plexus and reducing bowel motility. This increases intestinal transit time facilitating water reabsorption.

Notes

Acute diarrhoea

The priority in the management of acute diarrhoea, such as in gastroenteritis, is the prevention or reversal of fluid and electrolyte depletion. This is particularly important in infants, and in frail and elderly patients. Oral rehydration preparations may be used, or in more severe cases patients may be admitted to hospital and given intravenous fluid and electrolyte replacement.

Antimotility drugs

Antimotility drugs can relieve symptoms of acute diarrhoea by decreasing intestinal secretion and motility, prolonging intestinal transit time and facilitating water reabsorption (e.g. loperamide acting on opioid mu-receptors).

They may be used in the management of uncomplicated acute diarrhoea in adults but are not recommended for acute diarrhoea in young children.

Antimotility drugs are contraindicated in active ulcerative colitis, antibiotic-associated colitis and conditions where abdominal distension develops or where inhibition of peristalsis should be avoided.

Antispasmodics

Antispasmodics are occasionally of value in treating abdominal cramp associated with diarrhoea in adults but they should not be used for primary treatment.

Antispasmodics and antiemetics should be avoided in young children with gastroenteritis because they are rarely effective and have troublesome side effects.

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Pharmacology: Gastro, Fluids and MSK

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Proton pump inhibitors are indicated in all of the following EXCEPT for:

a Zollinger-Ellison syndrome

b Helicobacter pylori eradication

c Prophylaxis of NSAID-associated ulceration

d Post-endoscopic treatment of severe peptic ulcer bleeding

e Pancreatitis

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Pharmacology: Gastro, Fluids and MSK



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Proton pump inhibitors are indicated in all of the following EXCEPT for:

a) Zollinger-Ellison syndrome ✖
b) Helicobacter pylori eradication
c) Prophylaxis of NSAID-associated ulceration
d) Post-endoscopic treatment of severe peptic ulcer bleeding
e) Pancreatitis ✔

Answer

Proton pump inhibitors may be indicated for:

- treatment of gastric and duodenal ulcers
- eradication of Helicobacter pylori in combination with antibacterials
- treatment of dyspepsia and GORD
- post-endoscopic treatment of severe peptic ulcer bleeding (an intravenous high dose PPI reduces risk of rebleeding and the need for surgery)
- treatment and prevention of NSAID-associated ulcers
- cystic fibrosis (reduces degradation of pancreatic enzyme supplements)
- Zollinger-Ellison syndrome (controls excessive gastric acid secretion)

Notes

Mechanism of action

Proton pump inhibitors act by directly inhibiting the H^+/K^+ ATPase (proton) pump on gastric parietal cell luminal membranes and thus inhibiting acid secretion.

Indications

Proton pump inhibitors may be indicated for:

- treatment of gastric and duodenal ulcers
- eradication of Helicobacter pylori in combination with antibacterials
- treatment of dyspepsia and GORD
- post-endoscopic treatment of severe peptic ulcer bleeding (an intravenous high dose PPI reduces risk of rebleeding and the need for surgery)
- treatment and prevention of NSAID-associated ulcers
- cystic fibrosis (reduces degradation of pancreatic enzyme supplements)
- Zollinger-Ellison syndrome (controls excessive gastric acid secretion)

Cautions

Proton pump inhibitors:

- can increase the risk of fractures (particularly when used at high doses for over a year in the elderly or used in those at risk of osteoporosis)
- may increase the risk of gastrointestinal infections (including Clostridium difficile infection)
- may mask the symptoms of gastric cancer (in patients with 'alarm features', gastric malignancy should be ruled out before starting treatment)
- can cause false negative H. pylori test results (and should be stopped at least 2 weeks before testing)

Side effects

Common side effects (usually mild and reversible) include:

- abdominal pain
- constipation
- diarrhoea
- flatulence
- headache
- nausea
- vomiting
- dizziness
- skin rashes

Possible adverse effects of long-term PPI treatment:

- Hypomagnesaemia (measurement of serum magnesium concentrations should be considered before and during prolonged treatment with a PPI, especially when used with other drugs that cause hypomagnesaemia or with digoxin)
- Rebound acid hypersecretion and protracted dyspepsia after stopping prolonged treatment
- Interstitial nephritis
- Vitamin B12 deficiency

Administration

Omeprazole:

- H. pylori eradication regimen dose: 20 mg od
- Healing peptic ulcer: 20 mg od (40 mg od may be used in severe or recurrent cases)
- Healing NSAID-associated ulcer: 20 mg od
- Prophylaxis for NSAID-associated ulcer: 20 mg od
- Major peptic ulcer bleeding (following endoscopic treatment): Initially 80 mg, to be given over 40–60 minutes, then (by continuous intravenous infusion) 8 mg/hour for 72 hours, subsequent dose then changed to oral therapy

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Pharmacology: Gastro, Fluids and MSK

Question 43 of 49

Your patient has acute gout and you wish to prescribe colchicine. Which of the following is a contraindication to colchicine therapy:

a Heart failure

b Anticoagulant therapy

c Blood dyscrasias

d Diabetes mellitus

e Asthma

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Pharmacology: Gastro, Fluids and MSK



Question 43 of 49

Your patient has acute gout and you wish to prescribe colchicine. Which of the following is a contraindication to colchicine therapy:

a) **Heart failure** ✖
b) Anticoagulant therapy
c) Blood dyscrasias ✔
d) Diabetes mellitus
e) Asthma

Answer

Colchicine should be avoided in people with blood dyscrasias and bone marrow disease.

Notes

Gout is a disorder of purine metabolism characterised by a raised uric acid level in the blood (hyperuricaemia) and the deposition of urate crystals in joints and other tissues, such as soft connective tissues or the urinary tract.

NSAIDs

Acute attacks of gout are usually treated with an NSAID such as diclofenac, indometacin, or naproxen. Treatment should be started as soon as possible and continued until 48 hours after the attack has resolved. A PPI should be co-prescribed for gastric protection in people at high risk of gastrointestinal adverse events.

Colchicine

Colchicine is an alternative in patients in whom NSAIDs are contraindicated, not tolerated or ineffective. Although its use is limited by toxicity at higher doses, it is useful in patients with heart failure or those taking anticoagulants.

The dose is 500 micrograms 2 – 4 times a day until the symptoms are relieved or diarrhoea or vomiting occurs (max 6 mg per course and do not repeat treatment within 3 days).

Colchicine should be avoided in people with blood dyscrasias and bone marrow disease.

Common side effects of colchicine include:

- abdominal pain
- nausea and vomiting
- diarrhoea

Side effects are dose dependent and may be severe enough to limit treatment.

Corticosteroids

Oral or parenteral corticosteroids are an effective alternative in those who cannot tolerate or who are resistant to NSAIDs and colchicine. Intra-articular injection of a corticosteroid can be used occasionally in acute monoarticular gout.

Allopurinol

Allopurinol is not effective in treating an acute attack and may prolong it indefinitely if started during the acute episode, but should be continued during an acute attack if the patient is already established on long term therapy.

Long term treatment (prevention) of gout may be indicated in patients with frequent recurrence of acute attacks of gout, the presence of tophi, or signs of chronic gouty arthritis. Allopurinol reduces uric acid formation from purines and may be used to prevent further attacks of gout by correcting hyperuricaemia. The initiation of treatment may precipitate an acute attack, and should be covered with an NSAID or colchicine, continued for at least one month after the hyperuricaemia has been corrected.

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Pharmacology: Gastro, Fluids and MSK

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Regarding inflammatory bowel disease, acute mild to moderate disease of the rectum or rectosigmoid is typically treated initially with:

a Local aminosalicylate

b Local corticosteroid

c Oral aminosalicylate

d Oral steroid

e Intravenous steroid

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Pharmacology: Gastro, Fluids and MSK

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Regarding inflammatory bowel disease, acute mild to moderate disease of the rectum or rectosigmoid is typically treated initially with:

a) **Local aminosalicylate** ✔
b) Local corticosteroid
c) Oral aminosalicylate
d) Oral steroid
e) Intravenous steroid

Answer

Acute mild to moderate disease affecting the rectum (proctitis) or the rectosigmoid is treated initially with local application of an aminosalicylate; alternatively, a local corticosteroid can be used but it is less effective. A combination of a local aminosalicylate and a local corticosteroid can be used for proctitis that does not respond to a local aminosalicylate alone.

Notes

Inflammatory bowel disease

Aminosalicylates, corticosteroids and drugs that affect the immune response are used in the management of inflammatory bowel disease.

Patients who present with acute exacerbations of a known inflammatory bowel disease often require specialist advice and input.

Management of acute exacerbations

- Acute mild to moderate disease affecting the rectum (proctitis) or the rectosigmoid is treated initially with local application of an aminosalicylate; alternatively, a local corticosteroid can be used but it is less effective. A combination of a local aminosalicylate and a local corticosteroid can be used for proctitis that does not respond to a local aminosalicylate alone.
- Diffuse inflammatory bowel disease or disease that does not respond to local therapy requires oral treatment:
  - Mild disease affecting the proximal colon can be treated with an oral aminosalicylate alone; a combination of a local and an oral aminosalicylate can be used in proctitis or distal colitis.
  - Refractory or moderate inflammatory bowel disease usually requires adjunctive use of an oral corticosteroid such as prednisolone for 4 – 8 weeks.
  - Severe inflammatory bowel disease or disease that is not responding to an oral corticosteroid requires hospital admission and treatment with an intravenous corticosteroid (such as hydrocortisone or methylprednisolone); other therapy may include intravenous fluid and electrolyte replacement, and possibly parenteral nutrition. Specialist supervision is required for patients who fail to respond adequately to these measures.

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Pharmacology: Gastro, Fluids and MSK

Question 45 of 49

Regarding antimuscarinic antispasmodics, which of the following statements is CORRECT:

a They are of particular use for treating symptoms in gastroenteritis in children.

b Hyoscine butylbromide is a very effective antispasmodic.

c Antimuscarinics relax smooth muscle via blockage of nicotinic receptors.

d They are contraindicated in paralytic ileus.

e Antimuscarinics should be used with caution in patients with asthma due to thickening of bronchial secretions.

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Pharmacology: Gastro, Fluids and MSK



Question 45 of 49

Regarding antimuscarinic antispasmodics, which of the following statements is CORRECT:

a) **They are of particular use for treating symptoms in gastroenteritis in children.** ✖

b) Hyoscine butylbromide is a very effective antispasmodic.

c) Antimuscarinics relax smooth muscle via blockage of nicotinic receptors.

d) They are contraindicated in paralytic ileus. ✔

e)

Antimuscarinics should be used with caution in patients with asthma due to thickening of bronchial secretions.

Answer

Antimuscarinics are contraindicated in paralytic ileus. Antimuscarinics reduce intestinal motility by blocking muscarinic acetylcholine receptors and relaxing smooth muscle. Hyoscine butylbromide is advocated as a gastrointestinal antispasmodic, but it is poorly absorbed and thus has limited clinical utility. Antimuscarinics cause a reduction in bronchial secretions (they can be used to this effect in palliative patients). Antispasmodics are occasionally of value in treating abdominal cramp associated with diarrhoea but they should not be used for primary treatment. Antispasmodics should be avoided in young children with gastroenteritis because they are rarely effective and have troublesome side effects.

Notes

Antimuscarinics

Antimuscarinics reduce intestinal motility (by acting on acetylcholine receptors and relaxing smooth muscle) and can be used as antispasmodics for the management of irritable bowel syndrome. Hyoscine butylbromide is advocated as a gastrointestinal antispasmodic, but it is poorly absorbed and thus has limited clinical utility.

Other indications for antimuscarinic drugs include arrhythmias, asthma and airways disease, motion sickness, parkinsonism, urinary incontinence, mydriasis and cycloplegia, premedication, and as an antidote to organophosphate poisoning.

Cautions

Antimuscarinics should be used with caution in:

- acute myocardial infarction
- arrhythmias (may be worsened)
- autonomic neuropathy
- cardiac insufficiency (due to association with tachycardia)
- cardiac surgery (due to association with tachycardia)
- children (increased risk of side effects)
- conditions characterised by tachycardia
- congestive heart failure (may be worsened)
- coronary artery disease (may be worsened)
- diarrhoea
- elderly (especially if frail)
- gastro-oesophageal reflux disease
- hiatus hernia with reflux oesophagitis
- hypertension
- hyperthyroidism (due to association with tachycardia)
- individuals susceptible to angle-closure glaucoma
- prostatic hyperplasia
- pyrexia
- ulcerative colitis

Contraindications

Antimuscarinics are contraindicated in:

- gastrointestinal obstruction
- intestinal atony
- myasthenia gravis
- paralytic ileus
- prostatic enlargement
- pyloric stenosis
- severe ulcerative colitis
- significant bladder outflow obstruction
- toxic megacolon
- urinary retention

Side effects

Many drugs have antimuscarinic effects; concomitant use of two or more such drugs can increase side effects such as dry mouth, urine retention, and constipation.

Side effects of antimuscarinics include:

- dilation of pupils with loss of accommodation (cycloplegia) and photophobia resulting in blurred vision ("Blind as a bat")
- dry mouth, eyes and skin ("Dry as a bone")
- elevated temperature ("Hot as a hare")
- skin flushing ("Red as a beet")
- confusion or agitation particularly in the elderly ("Mad as a hatter")
- reduced bronchial secretions
- transient bradycardia followed by tachycardia, palpitation and arrhythmias
- urinary retention
- constipation

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Pharmacology: Gastro, Fluids and MSK

Question 46 of 49

Normal saline (sodium chloride 0.9%) contains which of the following:

a Sodium and chloride

b Sodium, chloride and bicarbonate

c Sodium, chloride and potassium

d Sodium, chloride and calcium

e Sodium chloride, bicarbonate and calcium

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Pharmacology: Gastro, Fluids and MSK



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Normal saline (sodium chloride 0.9%) contains which of the following:

a) **Sodium and chloride** ✔
b) Sodium, chloride and bicarbonate
c) Sodium, chloride and potassium
d) Sodium, chloride and calcium
e) Sodium chloride, bicarbonate and calcium

Answer

Normal saline (sodium chloride 0.9%) contains:

- Na^+ 150 mmol/L
- Cl^- 150 mmol/L

Notes

Solutions of electrolytes are given intravenously to meet normal fluid and electrolyte requirements or to replenish substantial deficits or continuing losses when the patient is nauseated or vomiting and is unable to take adequate amounts by mouth. When intravenous administration is not possible, fluid (as sodium chloride 0.9% or glucose 5%) can also be given by subcutaneous infusion.

Sodium chloride

Normal saline (sodium chloride 0.9%) contains:

- Na^+ 150 mmol/L
- Cl^- 150 mmol/L

Sodium chloride in isotonic solution (0.9%) provides the most important extracellular ions in near physiological concentrations and is indicated in sodium depletion which can arise from such conditions as gastroenteritis, diabetic ketoacidosis, ileus, and ascites.

Chronic hyponatraemia arising from inappropriate secretion of antidiuretic hormone should ideally be corrected by fluid restriction. If sodium chloride is required for acute or chronic hyponatraemia, regardless of the cause, the deficit should be corrected slowly to avoid the risk of osmotic demyelination syndrome and the rise in plasma sodium concentration should not exceed 10 mmol/L in 24 hours. In severe hyponatraemia, sodium chloride 1.8 % may be used cautiously.

Hartmann's solution

Hartmann's solution (compound sodium lactate) can be used instead of isotonic sodium chloride solution during or after surgery, or in the initial management of the injured or wounded; it may reduce the risk of hyperchloraemic acidosis.

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
- HCO_3^- 29 mmol/L
- Cl^- 111 mmol/L
- Ca^{2+} 2 mmol/L

Glucose

Glucose solutions (5%) are used mainly to replace water deficit. Average water requirements in a healthy adult are 1.5 to 2.5 litres daily and this is needed to balance unavoidable losses of water through the skin and lungs and to provide sufficient for urinary excretion. Dehydration may occur when these losses are not compensated for by intake e.g. in coma, or in the elderly. Excessive loss of water without loss of electrolyte is uncommon, occurring in fevers, hyperthyroidism, diabetes insipidus and hypercalcaemia.

Glucose solutions are also used to correct and prevent hypoglycaemia and to provide a source of energy in those too ill to be fed adequately by mouth.

Glucose solutions are also given in regimens with calcium and insulin for the emergency management of hyperkalaemia. They are also given after correction of hyperglycaemia during treatment of diabetic ketoacidosis, when they must be accompanied by continuing insulin infusion.

Potassium chloride mixtures

Potassium chloride with sodium chloride intravenous infusion is the initial treatment for the correction of severe hypokalaemia and when sufficient potassium cannot be taken by mouth. Repeated measurement of plasma-potassium concentration is necessary to avoid the development of hyperkalaemia, particularly in renal impairment. Initial potassium replacement therapy should not involve glucose infusions, because glucose may cause a further decrease in the plasma-potassium concentration.

Sodium bicarbonate

Sodium bicarbonate is used to control severe metabolic acidosis (pH < 7.1) particularly that caused by loss of bicarbonate (as in renal tubular acidosis or from excessive gastrointestinal losses). Mild metabolic acidosis associated with volume depletion should first be managed by appropriate fluid replacement because acidosis usually resolves as tissue and renal perfusion are restored.

In more severe metabolic acidosis or when the acidosis remains unresponsive to correction of anoxia or hypovolaemia, sodium bicarbonate (1.26%) can be infused over 3 – 4 hours with plasma-pH and electrolyte monitoring. In severe shock, for example in cardiac arrest, metabolic acidosis can develop without sodium or volume depletion; in these circumstances sodium bicarbonate is best given as a small volume of hypertonic solution, such as 50 mL of 8.4% solution intravenously.

Albumin

Albumin solutions, prepared from whole blood, contain soluble proteins and electrolytes but no clotting factors, blood group antibodies, or plasma cholinesterases thus may be given without regard to the recipient's blood group.

Albumin is usually used after the acute phase of illness to correct a plasma-volume deficit; hypoalbuminaemia itself is not an appropriate indication and the use of albumin solution in acute plasma or blood loss is wasteful where plasma substitutes should be used instead.

Concentrated albumin solution (20%) can be used under specialist supervision in patients with an intravascular fluid deficit and oedema because of interstitial fluid overload, to restore intravascular plasma volume with less exacerbation of the salt and water overload than isotonic solutions. Concentrated albumin solution may also be used to obtain a diuresis in hypoalbuminaemic patients (e.g. in hepatic cirrhosis).

Plasma substitutes

Dextran and gelatin may be used at the outset to expand and maintain blood volume in shock arising from conditions such as burns or septicaemia; they may also be used as an immediate short-term measure to treat haemorrhage until blood is available. Dextran and gelatin are rarely needed when shock is due to sodium and water depletion because, in these circumstances, the shock responds to water and electrolyte repletion.

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Pharmacology: Gastro, Fluids and MSK

Question 47 of 49

A patient presents to ED having recently been started on colchicine therapy for acute gout. Which of the following is the most common side effect that he is likely to complain of:

a Photosensitivity

b Diarrhoea

c Ankle swelling

d Rash

e Blurred vision

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Pharmacology: Gastro, Fluids and MSK



Question 47 of 49

A patient presents to ED having recently been started on colchicine therapy for acute gout. Which of the following is the most common side effect that he is likely to complain of:

a) **Photosensitivity** ✗
b) Diarrhoea ✓
c) Ankle swelling
d) Rash
e) Blurred vision

Answer

The most common side effect of colchicine is gastrointestinal upset (nausea, vomiting, diarrhoea and abdominal pain). Side effects are dose dependent and may be severe enough to limit treatment.

Notes

Gout is a disorder of purine metabolism characterised by a raised uric acid level in the blood (hyperuricaemia) and the deposition of urate crystals in joints and other tissues, such as soft connective tissues or the urinary tract.

NSAIDs

Acute attacks of gout are usually treated with an NSAID such as diclofenac, indometacin, or naproxen. Treatment should be started as soon as possible and continued until 48 hours after the attack has resolved. A PPI should be co-prescribed for gastric protection in people at high risk of gastrointestinal adverse events.

Colchicine

Colchicine is an alternative in patients in whom NSAIDs are contraindicated, not tolerated or ineffective. Although its use is limited by toxicity at higher doses, it is useful in patients with heart failure or those taking anticoagulants.

The dose is 500 micrograms 2 – 4 times a day until the symptoms are relieved or diarrhoea or vomiting occurs (max 6 mg per course and do not repeat treatment within 3 days).

Colchicine should be avoided in people with blood dyscrasias and bone marrow disease.

Common side effects of colchicine include:

- abdominal pain
- nausea and vomiting
- diarrhoea

Side effects are dose dependent and may be severe enough to limit treatment.

Corticosteroids

Oral or parenteral corticosteroids are an effective alternative in those who cannot tolerate or who are resistant to NSAIDs and colchicine. Intra-articular injection of a corticosteroid can be used occasionally in acute monoarticular gout.

Allopurinol

Allopurinol is not effective in treating an acute attack and may prolong it indefinitely if started during the acute episode, but should be continued during an acute attack if the patient is already established on long term therapy.

Long term treatment (prevention) of gout may be indicated in patients with frequent recurrence of acute attacks of gout, the presence of tophi, or signs of chronic gouty arthritis. Allopurinol reduces uric acid formation from purines and may be used to prevent further attacks of gout by correcting hyperuricaemia. The initiation of treatment may precipitate an acute attack, and should be covered with an NSAID or colchicine, continued for at least one month after the hyperuricaemia has been corrected.

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Pharmacology: Gastro, Fluids and MSK

Question 48 of 49

Normal saline (sodium chloride 0.9%) contains how much chloride:

a 131 mmol/L

b 150 mmol/L

c 111 mmol/L

d 170 mmol/L

e 126 mmol/L

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Pharmacology: Gastro, Fluids and MSK



Question 48 of 49

Normal saline (sodium chloride 0.9%) contains how much chloride:

a) 131 mmol/L
b) 150 mmol/L ✔
c) 111 mmol/L
d) 170 mmol/L
e) **126 mmol/L** ✖

Answer

Normal saline (sodium chloride 0.9%) contains:

- Na^+ 150 mmol/L
- Cl^- 150 mmol/L

Notes

Solutions of electrolytes are given intravenously to meet normal fluid and electrolyte requirements or to replenish substantial deficits or continuing losses when the patient is nauseated or vomiting and is unable to take adequate amounts by mouth. When intravenous administration is not possible, fluid (as sodium chloride 0.9% or glucose 5%) can also be given by subcutaneous infusion.

Sodium chloride

Normal saline (sodium chloride 0.9%) contains:

- Na^+ 150 mmol/L
- Cl^- 150 mmol/L

Sodium chloride in isotonic solution (0.9%) provides the most important extracellular ions in near physiological concentrations and is indicated in sodium depletion which can arise from such conditions as gastroenteritis, diabetic ketoacidosis, ileus, and ascites.

Chronic hyponatraemia arising from inappropriate secretion of antidiuretic hormone should ideally be corrected by fluid restriction. If sodium chloride is required for acute or chronic hyponatraemia, regardless of the cause, the deficit should be corrected slowly to avoid the risk of osmotic demyelination syndrome and the rise in plasma sodium concentration should not exceed 10 mmol/L in 24 hours. In severe hyponatraemia, sodium chloride 1.8 % may be used cautiously.

Hartmann's solution

Hartmann's solution (compound sodium lactate) can be used instead of isotonic sodium chloride solution during or after surgery, or in the initial management of the injured or wounded; it may reduce the risk of hyperchloraemic acidosis.

Hartmann's solution contains:

- Na^+ 131 mmol/L
- K^+ 5 mmol/L
- HCO_3^- 29 mmol/L
- Cl^- 111 mmol/L
- Ca^{2+} 2 mmol/L

Glucose

Glucose solutions (5%) are used mainly to replace water deficit. Average water requirements in a healthy adult are 1.5 to 2.5 litres daily and this is needed to balance unavoidable losses of water through the skin and lungs and to provide sufficient for urinary excretion. Dehydration may occur when these losses are not compensated for by intake e.g. in coma, or in the elderly. Excessive loss of water without loss of electrolyte is uncommon, occurring in fevers, hyperthyroidism, diabetes insipidus and hypercalcaemia.

Glucose solutions are also used to correct and prevent hypoglycaemia and to provide a source of energy in those too ill to be fed adequately by mouth.

Glucose solutions are also given in regimens with calcium and insulin for the emergency management of hyperkalaemia. They are also given after correction of hyperglycaemia during treatment of diabetic ketoacidosis, when they must be accompanied by continuing insulin infusion.

Potassium chloride mixtures

Potassium chloride with sodium chloride intravenous infusion is the initial treatment for the correction of severe hypokalaemia and when sufficient potassium cannot be taken by mouth. Repeated measurement of plasma-potassium concentration is necessary to avoid the development of hyperkalaemia, particularly in renal impairment. Initial potassium replacement therapy should not involve glucose infusions, because glucose may cause a further decrease in the plasma-potassium concentration.

Sodium bicarbonate

Sodium bicarbonate is used to control severe metabolic acidosis (pH < 7.1) particularly that caused by loss of bicarbonate (as in renal tubular acidosis or from excessive gastrointestinal losses). Mild metabolic acidosis associated with volume depletion should first be managed by appropriate fluid replacement because acidosis usually resolves as tissue and renal perfusion are restored.

In more severe metabolic acidosis or when the acidosis remains unresponsive to correction of anoxia or hypovolaemia, sodium bicarbonate (1.26%) can be infused over 3 – 4 hours with plasma-pH and electrolyte monitoring. In severe shock, for example in cardiac arrest, metabolic acidosis can develop without sodium or volume depletion; in these circumstances sodium bicarbonate is best given as a small volume of hypertonic solution, such as 50 mL of 8.4% solution intravenously.

Albumin

Albumin solutions, prepared from whole blood, contain soluble proteins and electrolytes but no clotting factors, blood group antibodies, or plasma cholinesterases thus may be given without regard to the recipient's blood group.

Albumin is usually used after the acute phase of illness to correct a plasma-volume deficit; hypoalbuminaemia itself is not an appropriate indication and the use of albumin solution in acute plasma or blood loss is wasteful where plasma substitutes should be used instead.

Concentrated albumin solution (20%) can be used under specialist supervision in patients with an intravascular fluid deficit and oedema because of interstitial fluid overload, to restore intravascular plasma volume with less exacerbation of the salt and water overload than isotonic solutions. Concentrated albumin solution may also be used to obtain a diuresis in hypoalbuminaemic patients (e.g. in hepatic cirrhosis).

Plasma substitutes

Dextran and gelatin may be used at the outset to expand and maintain blood volume in shock arising from conditions such as burns or septicaemia; they may also be used as an immediate short-term measure to treat haemorrhage until blood is available. Dextran and gelatin are rarely needed when shock is due to sodium and water depletion because, in these circumstances, the shock responds to water and electrolyte repletion.

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Pharmacology: Gastro, Fluids and MSK

Question 49 of 49

Which of the following best describes the therapeutic effect of calcium gluconate when used in treatment for hyperkalaemia:

a Promotes urinary potassium excretion

b Reduces cardiac cell membrane excitability

c Corrects acidosis and thus promotes intracellular uptake of K+

d Ion-exchange resin that exchanges sodium for potassium as it passes through the intestine

e Increases intracellular uptake of K+ via Na-K ATP pump

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Pharmacology: Gastro, Fluids and MSK

Question 49 of 49

Which of the following best describes the therapeutic effect of calcium gluconate when used in treatment for hyperkalaemia:

a) **Promotes urinary potassium excretion** ✗
b) Reduces cardiac cell membrane excitability ✓
c) Corrects acidosis and thus promotes intracellular uptake of K+
d) Ion-exchange resin that exchanges sodium for potassium as it passes through the intestine
e) Increases intracellular uptake of K+ via Na-K ATP pump

Answer

Calcium resonium is an ion-exchange resin that exchanges sodium for potassium as it passes through the intestine, leading to excretion of potassium from the body. Salbutamol and insulin act to increase intracellular uptake of K+ via Na-K ATP pump. Sodium bicarbonate acts to correct acidosis and thus promotes intracellular uptake of K+. Calcium gluconate acts to protect the cardiac membrane and has no effect on serum K+ levels.

Notes

Acute severe hyperkalaemia is characterised by a plasma-potassium concentration above 6.5 mmol/litre or the presence of ECG changes. ECG changes observed in hyperkalaemia may include tall peaked T waves, flattening or loss of P waves, broadening of QRS complexes, PR prolongation and bradycardia.

Emergency management

1. Reduce cardiac cell membrane excitability:
   - **10 ml calcium gluconate 10% by slow intravenous injection over 5 minutes**
     - ECG changes should improve within 1 – 3 minutes, but only has a transient effect of about 30 minutes
     - If there is no improvement, a further 10 ml should be given every 5 – 10 minutes until the ECG normalises (max 3 doses in total)
     - If the patient is taking digoxin, the calcium gluconate should be given slowly (mixed with 100 ml 5% glucose and given over 20 minutes) as rapid calcium administration may precipitate myocardial digoxin toxicity
2. Transcellular shift from extracellular to intracellular space:
   - **Intravenous infusion of insulin-glucose given as 10 units of Actrapid added to 50 ml of glucose 50% infused over 30 minutes**
     - Potassium should decrease by about 0.6 – 1.0 mmol/L in 15 minutes and the reduction lasts for about 60 minutes
     - This is repeated if necessary or a continuous infusion initiated
   - **Nebulised salbutamol 10 mg**
     - Reduces potassium by about 0.5 – 1.0 mmol/L in 15 – 30 minutes and lasts for about two hours
     - Should be used with caution in patients with ischaemic heart disease and history of cardiac arrhythmias
     - Response is attenuated in patients on beta-blockers and digoxin
   - The correction of causal or compounding acidosis with sodium bicarbonate infusion should be considered (if pH < 7.2)
     - Correction of acidosis promotes intracellular uptake of K+
3. Reduce total body potassium:
   - Any potassium supplements should be stopped, as well as any drugs that might precipitate hyperkalaemia (e.g. digoxin, beta-blockers)
   - Consider low potassium diet
   - Urinary potassium loss should be promoted with the use of appropriate fluids or diuretics to maintain good urine output
   - **Calcium resonium 15 g PO TDS**
     - Ion-exchange resin that exchanges sodium for potassium as it passes through the intestine
     - Should be given with regular laxatives to prevent constipation
     - Each gram removes approximately 1 mmol/L of potassium but onset is slow, taking 2 – 6 hours or longer
     - Contraindicated in patients with pre-existing hypercalcaemia

If the patient does not respond to the above measures dialysis will be required. DIalysis is likely to be needed if potassium is very high (> 7.5 mmol/L), if the patient is oligo/anuric, if the patient is already on long term dialysis or if the patient has advanced CKD.

Treatment	Administration	Mechanism of Action	Time to Onset of Action	Duration of Action	Achievable reduction of serum K+
Calcium resonium	15 g PO TDS	Ion-exchange resin that exchanges sodium for potassium as it passes through intestine	2 – 6 hours or longer	4 – 6 hours	Unknown
Calcium gluconate	10 ml calcium gluconate 10% by IV injection over 5 mins	Antagonises cardiac membrane excitability	1 – 3 minutes	30 minutes	N/A
Insulin-glucose infusion	10 units Actrapid in 50 ml glucose 50% infused over 30 mins	Increases intracellular uptake of K+ via Na-K ATP pump	Within 15 minutes	60 minutes	0.6 – 1 mmol/L
Nebulised salbutamol	10 mg nebulised	Increases intracellular uptake of K+ via Na-K ATP pump	Within 30 minutes	1 – 3 hours	0.5 – 1 mmol/L
Sodium bicarbonate	1.4% 500 ml IV over 2 hours	Corrects acidosis and thus promotes intracellular uptake of K+	After 60 minutes	Unknown	Unknown

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